INTER-RELATIONS BETWEEN COGNITIVE FACTORS IN THE PREDICTION OF OUTCOME AMONG CHRONIC ALCOHOLICS

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by

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"Science must begin with myths and the criticism of myths." Karl R. Popper (1957, p.177)

INTRODUCTION

This thesis is concerned with the role of cognitive dysfunction and other psychological processes in mediating the treatment outcome of hospitalized alcoholics. The research involves identifying antecedent correlates of cognitive dysfunction measured during hospitalization, and determining the relationship of this dysfunction to therapy involvement and post-treatment functioning. A further concern lies in the identification of other factors (demographic, social, and psychological), that influence these dependent measures, and their importance in this respect, relative to that of cognitive dysfunction.

Essentially, the research design is a longitudinal outcome study which employs multivariate statistical tracking through time rather than active manipulation of one or more variables as is characteristic of experimental studies. No apology is necessary for the approach taken, it is a quasi-experiment in real time. This is an established paradigm in a number of different sciences. Like all scientific investigations, the research involves a search for order, an attempt to identify, describe, and use systematic covariation between events as a step towards the prediction of theoretically and, in this instance, clinically relevant outcomes. Although this approach may preclude the derivation of strong causal statements, it is nevertheless highly informative.

Additionally, it is equal to experimental investigations in its ability to disconfirm hypothesized relationships. In the investigation of the role of individual differences over time, the approach taken here provides a corrective to experimental designs which have been built on a statistical base that has encouraged an excessively situationalist perspective.

The playing down of individual differences in the genesis and treatment of psychological disorders has not only been a product of the experimental methodology our science has developed. It has been reinforced by the widespread acceptance of psychiatric classification in clinical research. Such classification involves the lumping together of diverse individuals into categories and the subsequent treatment of these categories, both clinically and experimentally, as if they were homogeneous groups. Within-cell variance has been considered a nuisance to be tolerated or removed by covariance analysis rather than considered to be of interest in its own right. The field of alcohol studies provides a good example of how important individual differences have been ignored. It is also an area where the shortcomings inherent in this approach have become increasingly evident in recent years.

Because the general field of alcohol studies is currently in a turmoil, with old assumptions being turned on their head and widely accepted categories broken apart, some time is spent in the first chapter reviewing these

general developments, developments which have a direct bearing on the topic of this thesis. Essentially, the situation is one where simplicity, albeit an illusory simplicity, is being replaced with complexity. The research conducted in this thesis recognizes this complexity and is but one of a number of recent attempts to bring order out of current confusion.

Later chapters review the literature relating cognitive dysfunction to alcoholism and the literature relevant to the other psychological measures included in the research design. A systems model indicating the hypothetical relationship of these processes to therapy outcome is then outlined, providing a framework for the subsequent sections describing the design, data analyses, and results. Path diagrams are also used to make explicit some of the findings. In the final section, the results are discussed in relation to earlier findings, and suggestions are made for future research.

"Today's science may well be tomorrow's Alchemy."

Mahoney (1978)

CHAPTER ONE

THE BROAD PERSPECTIVE

The Extent of Problem Drinking

In New Zealand as in many other societies, exact, quantitative assessment of the extent of alcohol problems is lacking. It is generally considered, however, that their magnitude is immense. Because the effects of alcohol abuse are mediated via a complex series of causal pathways within the social fabric, touching directly or indirectly on all aspects of social life, it is not surprizing that we lack precise information. We also lack accurate statistics on what may at first sight appear to be a more straightforward issue, the number of alcoholics in our communities. An important reason for this lies in the adeptness of many persons with alcohol problems not only in hiding their drinking excess from others, but also in denying the import of these excesses to their own physical, psychological, and social functioning. Indeed, some clinicians consider the development of these skills to be diagnostic of the alcoholic condition! Even more fundamental to this issue is the lack of a generally agreed-upon definition of what constitutes alcoholism or problem drinking.

Although we lack precise information on the extent of alcohol and alcohol-related problems, estimates have been made. For example, Frazer McDonald (1971), speaking of the New Zealand situation, provided the following

figures:

30 percent of male psychiatric admissions are due to alcoholism.

30 percent of acute surgical beds in general hospitals are occupied by people whose major contributing factor is alcohol.

one in fifteen adult social drinkers in New Zealand is or will be an alcoholic.

over 35,000 people in New Zealand are currently alcoholics.

over 200,000 people (out of our population of just over three million) have their lives seriously disrupted by the alcoholism of family members.

Such estimates must be interpreted with fairly large credibility intervals in mind. Nevertheless, estimates from a number of countries with similar per capita alcohol consumptions to ours, yield similar figures, attesting to the extent of this social problem.

Developments in Alcoholism Research

In recent years, the effects of excessive alcohol consumption upon individuals and society at large has become a major concern of Governments in many parts of the World, both Capitalist and Socialist. This concern has been backed with large-scale funding of alcoholism research and programmes designed to prevent and reduce the pathology incumbent upon alcohol abuse. These developments have led to a vast increase in our base of empirical knowledge. This knowledge, in the course of just a few years, has led to a questioning of virtually all of the traditional assumptions that have dominated

the alcoholism field. In the area of social and behavioural research, we have reached a cross-roads. To use Kuhn's (1970) terminology, it is a time of paradigmatic transition. For the researcher, the second generation of research has begun. For the clinician and his or her clients, new realities, with the promise of more effective treatments, are forming. For the moment however, there is a schism between the research developments and the every-day-world of public policy and treatment services.

The research described in this thesis is conceptually grounded in the "new perspective" - the emergent framework that challenges many of the traditional alcoholism beliefs. Its physical setting, in contrast, is that of a hospital treatment programme, dominated at the time by the combined philosophy of Alcoholics Anonymous and the medical disease model. Because of the dual setting of this project - one conceptual, one physical, let us briefly consider the development of the ideas that produced these divergent positions. In so doing, the current research will be given both an historical and a conceptual context.

Changing Attitudes Towards Alcohol Abuse

During the Nineteenth and early Twentieth century in this country, as in other nations of predominantly Western European descent, very large intakes of alcohol were customary. Indeed, in the pioneering decades of

Nineteenth century New Zealand, drunkeness was undoubtedly much more common than it is today. For example, in 1861, the 15,000 settlers in the Canterbury Province supported six breweries and imported over 13.5 litres of spirits, 31.5 of beer, and nearly 9 of wine per person. In 1847, in Auckland, there was one conviction for drunkeness for every eight persons (Sinclair , 1969).

Although heavy consumption of alcohol was accepted, particularly for males, beyond a certain point people became labelled as drunkards. These unfortunate people were the butt or scorn and ridicule. Their lot was a pitiful one - moving from place to place, these places often being prisons or primitive dosshouses. Drunkards were generally perceived as sinful or irresponsible. As such, they were treated harshly by the law. It was noted in the Auckland Times in 1845 that police were in the habit of beating drunks with their staffs, tying their wrists with cords, and dragging them through the streets. As sinners against God as well as the state, they were also seen as a concern of the Church. In addition to, or as an adjunct toward saving their souls, some churches provided facilities for their care. However, these bore little resemblance to the treatment programmes that exist today.

During the late Nineteenth century, along with the

development of a middle class conscience and the women's movement, temperance groups sprung up to combat the "demon" drink. These groups exerted pressure that led to restrictions on the sale and distribution of alcohol. In some countries they managed to pressure Governments to enforce a period of prohibition. They contributed little, however, to the treatment of those people who in more recent times would have been considered to be alcoholics.

In the 1920's, small numbers of individuals with alcohol-related problems began to group together to help themselves and to fight for more humane treatment by other members of society. This social movement began in the United States, although it later spread throughout the World. With the exception of a few religious and medical persons , this movement and people with alcohol problems generally, were neglected by the developing helping professions. Without the involvement of scientifically trained researchers or clinicians, these self-help groups groped for a conceptual framework to order their experiences and provide a tool for inducing social change. It was at this time that the concept of alcoholism was coined. Like "adolescence", another conceptual development of the Twentieth century, "alcoholism" entered the vocabulary of our culture and provided a semantic focus around which certain types of personal experience and social behaviour were organized. In other words, it became a part of social reality, increasingly

influencing both the way people perceived alcohol abuse and the policy of Government agencies toward people with alcohol problems.

The Unitary Model of Alcoholism

Pattison et al (1977), consider the major elements of the alcoholism model that developed during the 1920's and became refined throughout the ensuing decades, up until the 60's, to be as follows:

1. There is a unitary phenomenon which can be defined as alcoholism.
2. Alcoholics and prealcoholics are essentially different from nonalcoholics (like diabetics).
3. Alcoholics may sometimes experience a seemingly irresistible physical craving for alcohol.
4. Alcoholics gradually develop a process called "loss of control" over drinking, and possibly even an inability to stop drinking ("first drink, then drunk").
5. Alcoholism is a permanent and irreversible condition ("once an alcoholic, always an alcoholic").
6. Alcoholism is a progressive disease which follows an inexorable development through a distinct series of phases.

Following from this perspective of alcoholism as a single disease, a unitary clinical entity with a progression along a predictable continuum, the only viable treatment goal has been considered to be the fostering of "insight" (acceptance of the disease model) and abstinence.

Alcohol problems are not the only grouping of "problems

in living" that have come to be viewed as a unitary disease entity. A myriad of behavioural deviations (in the Sociological sense) have been pushed into similar conceptual categories - thence the diagnostic manual of the American Psychiatric Association and similar national medical bodies. Apart from facilitating the expansion of the medical profession, allowing it to subsume behavioural phenomena within its frame of reference, these conceptual developments have led to some increase in the tolerance and sympathy shown to people with psychological and relationship problems. This increased concern has also fostered the development of treatment centres and the infusion of sufficient financial resources to encourage research on a very large scale. With this encouragement, a wide range of academic and scientific disciplines have finally begun to focus their attention on alcohol problems. It has been this group that has challenged the traditional model.

Criticism of the Unitary Model

To document the wealth of empirical findings that have undermined each of the six major elements of the traditional model would take an entire volume. Indeed, Pattison et al (1977) have already provided this service, making repetition of the task redundant. Instead, a brief summary of this work, and some of the broader implications will be outlined.

On a philosophical basis, the strongest attack on the disease concept of alcoholism and behavioural deviance more generally, has come from Sociology and Radical or Anti-Psychiatry. Writers within the field of the Sociology of Deviance, particularly those who embrace the 'Social Construction of Reality paradigm' (Berger & Luckman, 1966; Buckner, 1971) and the labelling theorists (e.g. Schur, 1971), have provided alternative conceptual frameworks from which to view individual behavioural deviance.

These sociologists have argued, along with the radical psychiatrists (e.g. Szasz, 1972), that the disease model is a tool for medical imperialism within our society. By claiming intrapsychic or organic (disease) causation, this position takes attention away from the problems within the social structure, or for the traditional Marxists, the economic order, that these theorists see as the major determinant of psychological distress. Thus, the disease model is also viewed as politically reactionary as it diverts attention from social processes that are considered by them to require radical change.

Apart from these ideological arguments, the labelling theorists in particular have made an important contribution by focussing upon the behaviour of people and organizations who diagnose and 'process' various kinds of deviance. They have shown how the number of people who receive disease labels is influenced by social factors

operating both within wider society and particular treatment centres, as much as by any behaviours or properties intrinsic in those who become labelled. Buckner (1971) has referred to alcoholics as 'universal fillers'. They are so described because a number of studies have shown how a wide range of social agencies draw on this group when they have a shortage of more 'serious' or 'genuine' cases. In Buckner's (1971) words:

> The supply of down-and-out alcoholics is copious, available, obvious, degraded, in need of help, and constant. In every city they exist in sufficient numbers to fill the police officers' time, the jails, the courts, the psychiatric wards, the hospitals, the welfare agencies, and the gospel missions. Alcoholics steal enough and are enough danger to themselves to constitute a police problem, sick enough to constitute a health problem, destitute enough to constitute a welfare problem, and lost enough to constitute a religious problem. Alcoholics are God's gift to social control agencies (p. 246).

Although even labelling theorists do not manage to do without labels such as 'alcoholic', implying that there is indeed something 'out there' that intrinsically warrants this description, they do underline the point that the number of diagnosed alcoholics at any given time is influenced by the state of the social institution rather than merely constituting a reflection of the number of 'true' alcoholics. Actually, their work takes us further by showing that the criteria for making a diagnosis can also change if there is pressure on an agency to show that it is working to capacity (e.g. to prevent cuts in funding or staff levels). Thus, not only

does their work provide a caution to the researcher who takes hospital records to reflect the extent of alcohol problems in the community, it also shows there is a need to recognize that the types of people who become diagnosed as 'alcoholics' will fluctuate according to characteristics of the labelling agency itself.

Another area of inquiry that has long been at odds with mainstream theories about alcoholism is the field of epidemiology. Epidemiological research has consistently linked prevalence of alcoholism with per capita consumption of alcohol. When per capita consumption rises, so do all of the rates of alcohol-related problems. These measures include those that are less influenced by organizational processes described in the previous section, for example, deaths from cirrhosis of the liver. The consistency of this relationship implies that the number of people diagnosed as suffering from alcoholism (according to disease theory, a permanent condition) actually rises and falls in accordance with the quantity of alcohol consumed by the total population. Whereas the traditional model locates the cause of alcoholism within the body or psyche of the individual, epidemiology suggests that the cause lies not so much in the drinker as in the drink.

An enormous amount of empirical investigation has addressed elements one and two of the 'traditional'

alcoholism model outlined on page ten - in an effort to find predisposing physical or personality factors unique to alcoholics. Regarding constitutional factors, a review of this literature up until 1971, concluded that no factors had yet been identified as strong causative elements in alcoholism (Kissen & Bergleiter; 1971, 1972). There is a need, however, to qualify this conclusion in the light of recent longitudinal research involving the adopted children of alcoholic biological parents (Bohman, 1978; Rutstein & Veech, 1978; Winokour, 1976). This group of investigations provides support for the view that genetic factors contribute to susceptibility to develop alcohol problems, particularly the most severe patterns. There is also some support for the view that at least a part of this genetic influence may be mediated via the hyperactive child syndrome which has in turn been related to the so-called sociopathic adult personality (Goodwin et al, 1975). However, although there is support for genetic causation, the majority of the dependent variable variance remains unaccounted for.

With regard to the search for a personality structure unique to alcoholics, a recent review concluded, "alcoholics are different in so many ways that it makes no difference" (Keller, 1972, p. 1147). In other words, so-called alcoholics are a very diverse group in terms of both physiology and psychology, overlapping with other groups on all criteria studied, except alcohol consumption. Thus, the causation of alcohol problems remains poorly

understood. From the hundreds of studies that have approached this issue, all that can be said with some certainty is that severe problem drinking appears to have a multi-causal origin, involving the interaction of a great many social, psychological, and physical factors. Given this complexity, from the point of view of statistical probability, it is very unlikely that any two individuals would follow an identical path in the development of their drinking problem.

The conclusions stated in the last paragraph do not necessarily mean that the investigation of individual differences among labelled alcoholics is a pointless preoccupation. Quite the contrary. Variety within alcoholic populations is potentially of great significance. A number of studies have identified subpopulations within treatment samples that may provide a basis for more refined treatment approaches, tailored to the particular problems of a given group (Jacobson, 1976(a); Wanberg, Horn, & Foster, 1977). Although the disease model with its assumptions of homogeneity, suggests a common causation and a unitary set of treatment goals and methods, there exist a number of studies showing that response to various treatment modalities is in large part a function of sociodemographic variables (Bateman & Peterson, 1971; Pattison, Coe, & Doerr, 1973; Tomsovic, 1970).

Within the traditional model, the craving concept

provides the major explanatory statement to account for an important characteristic of problem drinkers - that they continue to drink, or resume drinking after a period of abstinence, even though this behaviour has frequently been associated with negative consequences. Both the psychodynamic explanation for this (that it represents a death wish) and the craving notion, contain circular logic, making empirical testing of the assertions problematic. In the case of the craving hypothesis, the logic runs as follows: he drinks because he needs a drink and he needs a drink because he drinks. Mello (1972) and Pattison et al (1977) provide a more detailed account of the logical inadequacies and research findings related to this issue.

Recently, researchers have begun to look in some detail at the relapse process following periods of abstinence (either 'spontaneous remissions' or following treatment). In spite of the widespread attention given to the hypothetical craving mechanism by hospital staff and Alcoholics Anonymous, patients very seldom mention craving as a reason for relapse, either when they list the reasons themselves, or when they check off reasons that are given as possibilities in a structured questionnaire (Litman, Eiser, & Rawson, 1977; Ludwig, 1972; Marlatt, 1977). These studies find that rather than craving, the most common precipitants appear to be: (1) unpleasant affect, including social anxiety, frustration and difficulty expressing anger, and (2)

inability to resist social pressure in "at risk" situations.

"Loss of control", the fourth element in the unitary model, is used to explain why some problem drinkers continue to drink after they have started drinking; why they appear to have difficulty stopping after a few drinks. Pattison et al (1977) claim that over 70 studies refute Jellinek's (1946, 1960) assertion that alcoholics undergo some sort of biological transformation whereby from that point on, the intake of small amounts of alcohol triggers a full-scale process of physical dependence.

Experiments that are inconsistent with the loss of control hypothesis include those that have shown that when alcoholics are given free access to alcohol, they do not drink to oblivion, that subjects can initiate and terminate bouts, that abstinence can be 'purchased' from alcoholic subjects, and that alcoholics frequently taper off their drinking to avoid D.T.s (Mello & Mendelson, 1965, 1971, 1972; Oki, 1974). From their extensive literature review, Pattison et al (1977) claim that studies consistently show that within either a hospital or laboratory, the drinking of chronic alcoholics is largely under the control of environmental contingencies. From these considerations, it appears that a major shortcoming of the loss of control position has been that, until recently, it has diverted attention from factors that seem to have an important influence upon the post-

hospitalization drinking behaviour of alcoholics.

If the importance of these affective and situational factors in the relapse process can be verified, it seems probable that the effectiveness of treatment programmes could be improved by systematically teaching strategies to cope with these events. The optimal approach would very likely involve specifying each individual's particular deficits and tailoring a treatment programme for that individual's unique situation. For the precipitants already mentioned, a number of extant therapies have immediate relevance, for example, relaxation training, systematic desensitization, social skills and assertiveness training, and number of other focussed behavioural and cognitive interventions.

It has already been mentioned that a major assumption following from the traditional model is that alcoholics must achieve and maintain abstinence from alcohol in order to be rehabilitated. Contrary to this view, however, studies have demonstrated beyond doubt that a number of individuals, including those who meet even the strictest criteria for inclusion in the alcoholic category, can return to a reduced, non-problem alcohol intake either on their own or following traditional, abstinence-oriented treatment programmes (Miller & Caddy, 1977; Pattison, 1976). Pattison et al (1977) list 74 relevant and supportive references. Seventeen of these studies had controlled drinking as the treatment goal.

The majority of this latter group of studies employed some form of Behaviour Therapy, either as the primary intervention method or as part of a multimodal regime. In contrast to the findings of these studies, there is one report in the literature of a controlled drinking programme where a large percentage of patients initially managed to reduce their alcohol consumption but who, at a later followup, had all relapsed to pre-hospitalization 'alcoholic' levels (Ewing & Rouse, 1976).

Although the Ewing findings indicate that there is a need to conduct further longer term followup studies of controlled drinking programmes before we advocate them as the treatment of choice for alcoholics, the evidence is clearly overwhelmingly in favour of the view that for some alcoholics, sustained controlled drinking is a post-treatment reality. It would seem that the most profitable step now is to attempt to identify which individuals can realistically obtain, and more importantly, sustain, a controlled drinking outcome. In terms of the traditional model, these findings contradict the loss of control and permanence/irriversibility elements. They are also inconsistent with the final assumption, that alcoholism invariably progresses along a well defined 'downward' path unless it is arrested through the attainment of abstinence.

From the accumulated research, particularly that of the last ten years, it is evident that many of the widely

held "common truths" of the alcoholism field have been put to the test and found wanting. From the Logician's point of view, hypotheses that have consistently resulted in predictive failures when exposed to the risk of falsification, have greater logical implications for a theory than successful predictions (Popper, 1972). The failure of the major elements of the traditional model to survive falsification, coupled with a dearth of empirical corroboration, can only lead to the conclusion, to put it somewhat bluntly, that the view of alcoholism as a unitary, progressive disease process is in bad shape.

Alcoholism Research Today

From the foregoing, it is evident that the alcoholism field curently lacks a unifying conceptual framework that is capable of generating precise predictive statements. The field has also yielded only the first glimmerings of an answer to the fundamental question of why some people who drink develop alcohol problems when others do not. Additionally, the research literature has produced little by way of technology that has resulted in demonstrable improvement in the efficacy of treatment programmes. What the expanded data base has done, is to show that there is enormous complexity where simplicity was assumed. It suggests that like Don Quixote, we have been preoccupied with deceptively real fictions, which are in effect, merely crude images of our own making.

The complexity that is now acknowledged by researchers has led to disillusionment and confusion among many clinicians in the field. It is threatening to a large number of lay therapists, many of whom are ex-problem drinkers, committed to AA dogma for the maintenance of their own sobriety. Often, medical personnel are also threatened. In part this is because many of the empirical findings are contrary to their own experience and accumulated clinical lore. This is not surprizing, the very processes of perception have a built-in conservative bias - we tend to see what we expect to. A number of doctors and nurses, perhaps the majority, lack the training background necessary for the new treatment approaches. No doubt some recognize in the attempts to redefine alcoholism as something other than a disease, a direct challenge to their previously unquestioned right to dominate in the treatment field. From a scientific perspective, there is also widespread concern regarding some of the more extreme claims made for the efficacy of controlled drinking programmes. An impartial reading of this literature suggests that anything other than optimistic but very carefully qualified claims are, on the basis of findings to date, premature.

Thus, "alcohology" today is a troubled discipline. Unfortunately, although the clinical literature suggests a variation in individual reactions to threat, openess to new experience is not the most frequent response.

This does not mean that there are not some individuals who span both 'camps'. Nevertheless, two camps there are, with the medically oriented and A.A. supporters polarized toward one end and the academic researchers and behaviourally oriented clinicians polarized at the other. This situation is not unique to the field of alcohol problems. Pattison et al (1977) quote Ravetz (1971) who makes the following statement about the history of science:

> A folk science is a body of accepted knowledge whose function is not to provide a basis for further advance, but to offer comfort and reassurance to some body of believers... In an immature field of scientific development there is an inevitable conflict which occurs when the result of disciplined scientific enquiry conflicts with the beliefs of a folk science, usually a popular one which is also adopted by the established cultural organs of society. (p. 389)

While Ravetz's observation appears to be pertinent to the state of the alcoholism field today, it is important for those of us who consider ourselves to fit into the scientific or implicitly "enlightened" camp to be cautioned by the consideration that probably no theories within the Behavioural Sciences are ideologically neutral. In terms of relative power, prestige, and in some cases our very livelihoods, we have rather more than an 'academic' interest in the validity of the expanded framework. A further exercise in humility is a moments reflection on how often it is that one generation's 'heroes' provide the 'villians' for the next generation.

Some Implications of the New Perspective

Where do these various considerations leave us?

At this time, consistent with the traditional alcoholism beliefs, the majority of treatment programmes throughout the Western World continue to treat demonstrably heterogeneous populations as if they were a homogeneous group. This in itself would not be particularly disturbing but for the poor treatment outcomes associated with these programmes. Although the evaluation of treatment is a difficult task and the literature is plagued with methodological problems, it appears that one year post-treatment improvement rates (improved social functioning and abstinence or a greatly reduced intake) rarely exceed 30 to 40 percent (Orford, 1973; Pattison, 1966). Equally bleak is Orford and Edwards' (1978) report indicating that alcoholics given one interview session, did as well over a two-year followup period as a similar group that received long-term inpatient treatment in a conventional abstinence-oriented programme.

With regard to the provision of clinical services, there is a growing consensus that there is a need to identify clinically relevant individual differences within alcoholic populations and to determine how such differences interact with various forms of therapy and post-treatment variables to produce therapeutic changes.

To conduct this research, it will first be necessary to increase the range of treatment modalities available. By taking account of individual differences and tailoring treatment to either subgroups of patients who are alike on some prognostically relevant dimension, or to individuals, there is a reasonable expectation that treatment effectiveness might be enhanced. Although their names are more often linked with the development of the traditional disease model, Bowman and Jellinek (1941) considered that with the development of advanced research and statistical methods, the day might come when alcoholics could be differentiated and provisions made to treat these groups in different ways. That day now appears to be very close.

A question that some writers consider to be a crucial one is whether or not alcoholism should still be regarded as a disease process, or, for that matter, referred to as "alcoholism". Clearly, the accumulated research of the last decade indicates that if it is to be so regarded, the disease concept or concepts will have to be very different from that of the earlier unitary model. Essentially the question becomes, can a disease framework incorporate the demonstrated complexity of alcohol-related problems? Some clinicians (e.g. Keller, 1976) argue that it can. In presenting his case, Keller plays down the pathophysiological aspects that dominated

the unitary model and utilizes the more behavioural and social aspects. Because disease has become a complex, multi-level concept in our culture (Parsons, 1958), this extension and change of emphasis is legitimate. However, to some sociologists and anti-psychiatry theorists, herein lies its danger - its capacity to expand insidiously at the convenience of the medical profession to engulf ever larger segments of social reality!

Whether or not alcoholism in <u>all</u> of its manifestations can be considered a disease seems to boil down to a question of semantics. In describing this "disease", Keller claims that he <u>believes</u> that it does have a physical etiology but that whether or not this is ever established, its behavioural manifestation as a disablement is sufficient to warrent the disease label. Whether or not this is sufficient reason to regard something as a disease is surely a matter of opinion. Although, one can readily think of other "behavioural disablements", for example illiteracy due to inadequate education, that even Keller would be unlikely to classify as a disease.

At other points, Keller refers to alcoholism as a "learned response" and mentions other behavioural disablements (by his definition diseases) that can be removed by psychotherapy. Thus, although accepting the complexity indicated by the more recent literature, it

is hard to avoid the arguments of those social scientists who consider that this is a case of stretching the medical model to its ultimate limits. As theories are generally less productive at the extremes of their range of convenience, and because a number of non-disease models are addressed more directly to sections of this data and have already been fruitful in generating research, it could be expected that these alternative positions should, in the longrun, produce more by way of treatment technology to assist with alcohol problems. This is not an issue that can be settled by armchair discourse. It is something that future research and clinical practice will hopefully settle. The proof of the pudding is in the eating! To date, it has been argued by Pattison et al (1977) and others, that a major consequence of the disease conceptions has, in fact, been to hinder the widespread testing of such treatment alternatives.

With regard to the term alcoholism, it seems that as a shorthand label to describe a group of people with alcohol-related problems, it is as adequate as any other term and somewhat less clumsy to use. It will be used in this way in this thesis. It needs to be noted, however, that it is being employed simply as a general descriptive term for a heterogeneous population of people with a common problem in the overuse of alcohol, a problem that disrupts their physical, psychological, and social

wellbeing. It is recognized that their heterogeneity extends to the nature of their drinking patterns, their physical, psychological, and social characteristics. Its use does not imply acceptance of the unitary model, a conception that has in the past become almost synonomous with this term.

"O thou invisible spirit of wine! if thou hast no name to be known by, let us call thee devil! ...O God! that man should put an enemy in their mouths to steal away their brains!" Shakespear (Othello)

CHAPTER TWO

BRAIN DAMAGE AND COGNITIVE DYSFUNCTION IN CHRONIC ALCOHOLICS

Introduction

Prolonged and excessive use of alcohol is associated with the presence of a very wide range of medical disorders. Pathology can occur in any organ system and is due either to the direct toxic effect of alcohol on body tissues, or to other conditions that commonly accompany alcohol abuse, for example, dietary neglect. Indeed, the variety of pathology involved is such that medical professionals are wont to quip that to know alcoholism is to know medicine.

Of the medical disorders that accompany alcohol abuse, those in the neurological category are the ones of most direct relevance to behaviour and cognition. Acute neurologic disorders include convulsions, hallucinosis, and delirium tremens. These symptoms, and in the case of delirium tremens, symptom clusters, frequently accompany the withdrawal phase. Wernicke's encephalopathy also typically has an acute onset and is characterized by nystagmus, sixth-nerve palsy, ataxia, and a confusional state. With careful attention to diet, this syndrome clears. Frequently it gives way to Korsakoff's psychosis which, in contrast to Wernicke's syndrome, shows little or no reversibility. This condition is

marked by a severe memory disorder and, in the early stages, confabulation. Other aspects of intellect are little disturbed. Gait disturbance and polyneuropathy are also extremely common although both of these symptoms are also frequently found as sequelae of alcoholism in the absence of Korsakoff's syndrome. Coma and myopathy (muscle degeneration) can also accompany prolonged periods of drinking. Typically, both are reversible.

Apart from Korsakoff's syndrome, which is associated with atrophy of specific subcortical sites, there are two further neurological conditions that are associated with degeneration in known brain loci. Both are rare and usually terminal. One involves degeneration of the corpus callosum (Marchiafava-Bignami disease) and appears to be almost exclusively confined to middle aged or elderly Italian men whose alcohol consumption consists largely of wine (Packard, 1976). The other, central pontine myelinolysis, is usually associated with quadriplegia and leads to death within a few weeks of onset.

Although overshadowed in the neurological literature by the acute states and Korsakoff's psychosis, there are a number of references in the earlier literature, dating back to the 1800's, that describe a subgroup of alcoholics who develop a more global deterioration of psychological functioning in which memory loss is only one aspect of a

more general intellectual decline (Cutting, 1978a; Horvath, 1975). The terms "chronic alcoholic deterioration" and "alcoholic dementia" have been used to desribe this condition. Over recent decades these terms appear to have been dropped in diagnostic practice. Indeed, there is no specific category covering this condition in the eighth revision of the International Classification of Diseases (ICD-8), only the very general category of alcoholic psychosis unspecified (291.9). Horvath (1975) and Cutting (1978b) have shown that in clinical practice, this trend has been accompanied by a loosening of the criteria for inclusion within the Korsakoff category.

In Cutting's study, it was shown that a high proportion of patients diagnosed as Korsakoff's psychosis, upon closer examination, had more global intellectual deterioration with a gradual onset. This finding has been confirmed by the present author and others (Note 1). It was found that in a large urban New Zealand psychiatric hospital, twelve percent of the total hospital population carried a primary diagnosis of Korsakoff psychosis. However, from a careful review of the casenotes, psychological test results, and interviews with these patients, two independent assessors agreed that slightly less than a quarter of this group closely fitted the ICD-8 criteria for the Korsakoff diagnosis. The majority of the remaining patients had evidence of more general deterioration in intellect and personality, presumably due either to alcohol abuse and related personal and

dietary neglect, to age, or to both. This pattern of dementia was either the primary feature or appeared to be superimposed on the Korsakoff condition.

In summary, from the neurological and psychiatric literature, the major categories of chronic mental disorder that accompany alcohol abuse and are of sufficient severity to come to medical attention, include the following:

(1) Korsakoff's syndrome. This condition appears to follow an initial confusional state and is characterized by a severe short-term memory disorder in the absence of other major intellectual deficits. It has an acute onset following thiamine deficiency and is associated with damage to specific thalamic nuclei and other subcortical sites.

(2) Alcoholic dementia. This condition resembles Korsakoff's syndrome in certain respects and is often misdiagnosed as such. It differs in that its development is more gradual, it is less dependent on nutritional factors, typically is associated with a longer drinking history, and is characterized by a more general intellectual decline or dementia. Some degree of recovery is the rule rather than the exception. In contrast to the Korsakoff condition, the major locus of brain atrophy is considered to be cortical.

(3) Alcoholic dementia and Korsakoff's syndrome combined. From Horvath (1975), Cutting (1978b), and our own work, it appears that in a moderate percentage of

hospitalized alcoholics with diagnosed organic brain syndromes, the two conditions outlined above coexist.

In Horvarth's (1975) survey, 100 of 1,100 (9 percent) alcoholics seeking treatment were diagnosed as possessing a chronic organic brain syndrome (20 of the 100 were considered to be Korsakoffs). It is uncertain how representative this sample is of the entire alcoholic population. Nevertheless, it is clear that the vast majority of alcoholics do not display neurological pathology sufficient to be considered on clinical examination to have a diagnosable brain syndrome.

Although the majority (probably more than 90 percent) of individuals seeking help for alcohol problems do not have clinically diagnosable organic deterioration, in the last ten years, neurological, psychological, and more recently neuropsychological, investigations have strongly suggested that more subtle forms of brain damage and cognitive dysfunction accompany prolonged periods of alcohol abuse. In contrast to the categorical approach of the medical diagnosticians, these investigators have marshalled evidence to support the view that organic and psychological deterioration exists among alcoholics along a quantitative continuum. This continuum ranges from none, through so-called 'soft' neurological signs and neuropsychological dysfunction, to grade into the obvious deterioration of the chronic brain syndromes. It is considered that the alcoholic dementia syndrome

represents the clinical manifestation of this continuum near its extreme pathological end (Cutting, 1978b). The slow onset of this condition and its association with a lengthy history of excessive alcohol intake make it a more likely contender than the Korsakoff syndrome with its more acute onset, specific brain loci, and distinctive psychological sequelae.

An extensive and rapidly growing literature now exists within neurology, clinical and cognitive psychology, and neuropsychology, that bears directly on the nature and extent of 'organicity' and cognitive inefficiency in alcoholic patients. For the present however, this work remains largely ignored in orthodox accounts of alcoholism. Its implications for the so-called (and probably largely mythical) 'alcoholic personality' and treatment, remain unexplored.

In the remainder of this chapter, this literature will be summarized and the possible relevance of this work to the understanding of alcohol-related problems and their treatment will be discussed. Another large body of literature which has an indirect bearing on this issue is that which attempts to delineate the acute effects of alcohol on physiological and psychological functioning in human and animal subjects. This work will not be systematically covered here although it is recognized that in the future, a time may come when there will be much to be gained by exploring in detail the

parallels that appear to be emerging in the findings of these currently separate areas. In particular, the study of the chronic effects of prolonged alcohol abuse would very probably benefit from the conceptual fertility of cognitive psychology, a field upon which human studies of acute alcohol effects are now starting to draw heavily (for example, see Birnbaum & Parker, 1977).

Neurological Evidence of Brain Damage

As indicated in the previous section, for many years it has been known that excessive alcohol consumption is a precursor of several types of neuropathy. Typically, these conditions follow many years of alcohol abuse, show obvious signs of brain atrophy upon neurological examination, and result in readily observable psychological deficit. This section is concerned with evidence from the medical and biological sciences that relates to the question of whether or not organic brain pathology also exists, albeit to a lesser degree, in the vast majority of alcoholics who fail to show obvious signs of neurological impairment.

The most direct method of assessing organic brain pathology is post mortem examination. Over the years, a number of such investigations have been conducted with chronic alcoholics. Courville (1955) describes the anatomical picture that often emerges. He describes cortical atrophy most extensive in the frontal lobes but frequently widespread with ventricular enlargement.

Upon microscopic examination of the cortical tissue, cell loss was observed. A study by Lynch (1960), of chronic alcoholics with an adequate nutritional background and nonalcoholics of the same age and sex, found that in the alcoholics at post mortem, this cortical cell loss ranged between 10 - 40 percent of the total neuronal population. This loss was far in excess of that of the nonalcoholic controls. These and other studies (see Ron, 1977), have also indicated an increase in the non-neuronal glial cell population and architectural derangement of the cortical laminae. Atherosclerotic changes have also been observed. Although Courville (1955) considers that these changes are due to the toxic effects of alcohol, this conclusion is still somewhat speculative.

From the abovementioned post mortem studies, it appears that the presence of cortical atrophy, at the macro and microstructure levels, is characteristic of alcoholic patients. Although typically diffuse in nature, it often appears to have a frontal focus. In addition to these findings, there is a suggestion that in at least some alcoholics, deterioration in anterior regions of the brain includes damage to subcortical areas (Tarter, 1976). For example, Creutzfeldt (1928), found that frontal-diencephalic tracts were disrupted in some alcoholics. As this area is also implicated in the Korsakoff syndrome, it may be that there is some continuity with this disorder as well as the predominantly

cortically mediated brain syndromes?

Individuals who become available for post mortem examination form a non-random sample of alcoholics. In particular, they are typically elderly persons with long drinking histories. For this reason, the findings mentioned above cannot be generalized to the entire alcoholic population, especially to younger alcoholics and those with shorter drinking histories. Consequently, although suggestive of the type of brain damage that may be involved, these studies do not allow estimates of the incidence and distribution of extent of brain damage among alcoholics seeking treatment.

Two other methods have been used by medical researchers in the investigation of brain changes in alcoholics, the pneumoencephalogram (PEG) and the echoencephalograph. The former, which provides a means of visualizing the macrostructure of the cerebrum by the injection of air into the subarachnoid space, has been used most frequently. Unfortunately, few of these studies have included adequate controls. Of those that have, the most adequate would seem to be an investigation by Haug (1968). This researcher assessed a large number of alcoholics with the PEG during the second to fourth week after admission. The mean age of the group was 45 and their mean drinking history 15 years. Seventy-four percent of this group was considered to have cerebral atrophy. This compared with eight percent in a matched group of schizophrenics. Haug also found that length of

drinking history was positively and significantly correlated with degree of cerebral atrophy. Of the 21 alcoholic patients who were considered to have personality disturbance that affected "moral and social aspects more than intellectual faculties", 20 had cerebral atrophy. Because the control group in this study was more likely to include brain damaged subjects than a group drawn from a non-hospital population, the difference between these two groups is probably of greater significance than would be furnished by comparison with a randomly drawn group.

Although lacking a control group, another study that is worthy of note because younger and less deteriorated subjects were involved, is that of Brewer and Perrett (1971). These researchers excluded subjects over the age of 60 years as well as those with clear evidence (from clinical observation) of brain damage. The subjects were psychiatric patients who had a regular daily intake of 3 litres of beer or its equivalent. The mean age of the group was 50 years. Again the PEG was used. The authors considered that cortical atrophy was present in 91 percent of the sample. Only six percent were found to have a normal PEG. Within the group with cortical atrophy, frontal lobe involvement (judged by the width of the frontal sulci) was observed in 93 percent. Parietal atrophy was also noted in 70 percent of the brain damaged group, in the vast majority of cases, in association with frontal deterioration. It is worth noting that only some of the

subjects included in this study were considered to be alcoholics, many were regarded as "heavy social drinkers". It may be that as far as brain damage is concerned, alcohol is no respector of the alcoholic - non-alcoholic distinction that many upholders of the unitary model hold as sacred?

A further interesting report by Tumarkin et al (1955) is noted by Ron (1977). Here, seven young men (age range 25 - 38 years) were referred for assessment because of declining work performance within a military setting. Their average drinking history was 11 years and causes for brain damage other than alcoholism were ruled out. All showed clear evidence of cortical atrophy. This study indicates that brain damage occurs among some heavy drinkers when background and age make causes other than alcohol unlikely. Because all seven previously showed evidence of a superior work record, the results also suggest that this deterioration, at least in this instance, followed rather than preceded a history of heavy alcohol consumption.

In addition to the studies cited, Parsons (1977), in his review of the literature, located a further eleven studies of alcoholics that included either the PEG or echoencephalography. Overall, these studies implicate the presence of both cortical and subcortical (periventricular atrophy) in hospitalized alcoholics. The percentage of subjects showing evidence of atrophy

ranged between 50 to 100 percent. The lower percentages came from studies where subjects were selected at random from inpatient alcoholic populations. Where subjects were chosen for investigation because of the suggestion of organic involvement from clinical observation or other testing, the percentages fell within the 90 - 100 range. Parsons concluded that on the basis of the extant literature, it would be incorrect to consider that all alcoholics have demonstrable brain damage. Nevertheless, within an unselective alcohol treatment programme, such damage will exist in somewhere between 50 to 60 percent of patients. The specific nature of the atrophy found in these studies was very similar to the pattern of gross pathology indicated by the post mortem examinations of older alcoholics.

Apart from the slender evidence from Tumarkin et als' study, the literature does not directly bear on the question of whether the damage demonstrated antedates or follows alcohol abuse. However, the correlation between degree of atrophy and years drinking in one of the studies (Haug, 1968), taken in conjunction with Tumarkin et als' report, suggests that at least some of the observed atrophy post-dates alcohol abuse. However, this evidence is indirect. Longitudinal studies are required to settle this issue.

The literature also tells us little of the temporal course of demonstrated lesions. Are they

static, progressive, or reversible? Hopefully, the recent introduction of the EMI scanner, a non-obtrusive and rapid procedure for assessing gross pathology, will greatly assist in the task of addressing these questions in the next few years.

Intellectual and Cognitive Functioning in Alcoholics

Introduction

Given that the Central Nervous System and the brain in particular, provide the major organic substrate for human behaviour, particularly the so-called 'higher mental functions' of intelligence and cognition, it is to be expected that the presence of lesions and atrophy in this organ will influence these psychological processes. Related to this issue, we have already noted the presence of gross behavioural disturbance in the organic brain syndromes, both acute and chronic. In many alcoholics who do not evidence this degree of organic pathology, obvious psychological deficits in the areas of motor coordination, memory, and orientation, are clearly evident in everyday behaviour, particularly while they are drinking or during the first few weeks of abstinence. However, in the majority, these deficits are not so readily observed after the 'drying out' period. The major concern of this section lies in the identification of more subtle cognitive changes that persist beyond acute intoxication or withdrawal periods. Before we look at this, however, a few

comments will be made regarding the acute effects of alcohol on non-alcoholic subjects.

Acute effects of alcohol on psychological functioning

Although little investigated, alcohol has fairly obvious effects on anxiety, aggression, motivation, and the dynamics of interpersonal interaction, when ingested by non-alcoholic individuals. Many people report feeling less anxious, less inhibited, and bolder in a variety of interpersonal situations. These changes probably stem from a reduction in self critical or inhibitory mechanisms that typically regulate behaviour via anxiety-linked cognitive processes. It is for this reason that it is often quipped that the superego may be operationally defined as that part of the brain that is soluble in alcohol! In the light of this discussion, it is of interest that the leucotomy, a psychosurgical procedure whereby fibres connecting the frontal cortex to subcortical areas are sectioned, usually results in similar behaviour changes. It may well be that the motivation for some individuals to drink to excess arises from alcohol's capacity to induce this reduction in self-critical attitude. Given the frontal focus of much of the cortical damage found in chronic alcoholics, it may even be that this functional change becomes a more permanent structural change.

The most studied of the psychological effects of acute intoxication are disturbances to memory.

Although not apparent to most drinkers, experimental investigations have established that even moderate doses of alcohol in social drinkers produce measurable decrements in memory and learning capacities.

Although it is now recognized as somewhat of an oversimplification, two broad types of memory processes are generally recognized, primary and secondary memory. Primary memory is a short-term process that corresponds to the holding in 'mind', in conscious awareness, of images and percepts. Secondary memory is a longer-term group of processes, covering the removal of items from short-term memory, their placement into some form of longer-term storage, and their subsequent retrieval to primary memory.

It is secondary memory that is most impaired by intoxication. To illustrate this, let us consider the following list of words:

ram
toffee
cup
dog
wool
wombat
table
emu
love

Experiments have been conducted where similar lists have been presented to normal subjects, both sober and intoxicated. Both groups do equally well on the recall of the last few words when they are asked to list the words immediately after presentation. Intoxicated subjects however, do much worse on words earlier in the

list. If a delay, coupled with a distractor task to prevent rehearsal (e.g. counting backwards in fives), is interspersed between presentation and recall however, then intoxicated subjects also perform at a lower level than non-intoxicated subjects on the last words in the list. It has been from experiments like this that the conclusion has been reached that it is secondary rather than primary memory processes that are impaired by acute intoxication.

Many experiments have been conducted to further clarify the nature of this secondary memory impairment (see Birnbaum & Parker, 1977). One of the more consistent findings has been the demonstration that when intoxicated subjects are cued in certain ways (e.g. by presenting subjects with a long list of words including those that were already given for learning, or in the case of the word list on the previous page, providing semantic cues such as; "two of the words were Australian animals") then the difference between intoxicated and non-intoxicated subjects almost or completely disappears. From these findings, it has been concluded that intoxicated subjects suffer a reduction in available processing resources, with the result that they engage in fewer spontaneous encoding operations. This and other work has further indicated that this encoding deficit is particularly marked in more 'elaborate' or 'deep' (semantic) encoding mechanisms and that the disruption occurs at both input and retrieval levels.

The relevance of these changes to the genesis of alcohol problems has not been explored. It may be, as was postulated in relation to reduced self-critical attitude, that for some people, a reduction in cognitive efficiency is rewarding? An anecdote that might be relevant here comes from the author's recent experience in recruiting brain damaged alcoholic subjects for a trial of Hydergine, a drug that has been of some value in restoring certain areas of cognitive functioning in elderly patients with poor cerebral blood flow. On explaining to one very deteriorated patient that the drug might be able to help to restore her very deficient memory, she indicated, along with a string of obscenities, that this was the last thing she wanted. "I don't want to be able to remember!" she said.

Again, there is some evidence that the memory changes found in intoxicated normals, become chronic in some alcoholics. In particular, patients with Korsakoff's Psychosis, suffer from very severe disturbances to secondary memory. Research indicates that as with acutely intoxicated normals, Korsakoffs are particularly deficient in their capacity to spontaneously encode, particularly at semantic levels (Butters & Cermak, 1974; 1976). Although typically ignored in the literature addressing cognitive dysfunction in sober chronic alcoholics, those experiments with Korsakoffs that have included alcoholic controls, have consistently shown that in this area of memory functioning,

the alcoholics lie between normals and Korsakoffs in degree of impairment. Thus, although it is generally thought that the Korsakoff condition is an acute neurological illness with unique anatomical and psychological sequelae, these findings suggest that at least one major cognitive aspect of this condition is found in chronic alcoholics prior to its more fully developed manifestation in the Korsakoff. In other words, it appears that we have another area of functioning where there is a continuum of disturbance from early alcoholism through to an organic brain syndrome.

Cognitive and perceptual performance in sober alcoholics

Two major lines of research have addressed the issue of cognitive and perceptual functioning in alcoholics after the withdrawal period. One has occurred independently of the biological findings. This line of research has followed the psychological tradition of attempting to delineate the motor, perceptual, and cognitive behavioural performance of alcoholics and has in part been concerned with how such factors may have led to an individual becoming an alcoholic. It is this research that will be reviewed now. The second tradition has been more concerned with the investigation of psychological dysfunction in alcoholics and how these changes relate to the organic changes noted in the earlier sections of this chapter. The major focus

here has been to specify brain-behaviour relationships, to add to our knowledge of which brain structures mediate particular psychological functions, and to evaluate the possibility of inferring structural brain damage, either generally or in specific locations, from psychological test performance. This work will be summarized in the next section. Although these two literatures overlap, they have had somewhat distinct histories and have arrived at rather different explanations for the behaviours observed in alcoholics.

Intelligence. Many studies have compared samples of alcoholics with matched controls on standardized measures of intelligence. A variety of tests have been used, including the Terman-Merrill (e.g. Amark, 1951), the Wechsler-Bellevue (e.g. Fitzhugh et al, 1960, 1965), and the WAIS (e.g. Goldstein & Shelly, 1971). Of the numerous studies, those listed above are the more adequate methodologically. In all cases, where the overall intelligence quotient (IQ) is considered, no significant difference is found between alcoholics and normals. However, with the exception of the Fitzhugh et al studies, these and other investigations have found that when overall IQ is broken into its verbal and performance components, the performance scores are typically lower than verbal scores. This verbal-performance discrepancy is typically intermediate between nonpsychiatric and brain damaged (particularly those with a right hemisphere involvement) groups. Additionally,

although certain of the subtest scores consistently "hold up" (i.e. are of a similar magnitude to the scores of nonalcoholics of the same age and level of education), particularly the Information and Vocabulary subtests, all of the other subtests have in at least one study been shown to be depressed. Implicit in the last sentence is the further point that in terms of subtest patterns, there is considerable variation from one study to the next. Indeed, a review by Kleinknecht and Goldstein (1972) concluded that the only subtests consistently found to be impaired were Object Assembly and Digit Symbol. Although less consistent, a number of studies have also indicated lower performance on the Block Design subtest (Grassi, 1953; Jonsson et al, 1962; Claeson & Carlsson, 1970; Goldstein et al, 1970; Smith & Smith, 1977). Given these varied findings, and the small magnitude of the differences between alcoholics and nonalcoholics when they are demonstrated, recent reviewers (e.g. Ron, 1977; Tarter, 1976) are in agreement that traditional psychometric tests of intelligence have been of only limited value in elucidating the nature and extent of cognitive impairment in alcoholics. Before leaving the question of intellectual functioning in alcoholics however, let us consider more carefully what these tests are in fact measuring.

There is considerable controversy with respect to what intelligence is and how adequately various tests

measure it (see Savage, 1970). Nevertheless, in spite of these difficulties, many psychologists consider it to be an important dimension along which humans vary. Additionally, performance on such tests has been shown to predict behaviour in a variety of real-life situations. At a very general level, many would probably accept Wechsler's view that intelligence represents ".. the aggregate or global capacity of the individual to act purposefully, to think rationally, and to deal effectively with his environment ". Although, this very broad definition also admits elements that are more commonly considered to be aspects of emotionality or personality.

In practice, test results are typically expressed by an overall measure, the IQ, which presumably bears some relation to Wechsler's definition. Although often indexed in this way, there seems to be a consensus that intelligence is comprised of a somewhat fluid collection of somewhat distinct, although nonetheless overlapping, abilities or intelligences. Vernon makes this explicit in his definition of intelligence as "a cluster of high grade skills concerned with problem-solving". Skills include verbal ability, word fluency, inductive reasoning, number ability, and others - the number of abilities and the relative importance attached to each depending on the particular theorist. However, irrespective of the specific abilities identified, for any given individual, in most instances, there is considerable overlap (high inter-correlation) between different types

of abilities. It is widely considered that this overlap is accounted for by an underlying general intelligence (g). It is also considered that overall IQ, derived in most tests from a summation of subtest scores, bears some relation to the hypothetical construct g, and that it is for this reason that it is capable of predicting performance in other settings.

A number of theorists, from different starting points, have arrived at the conclusion that there is theoretical justification and practical utility in regarding g as having two separate components. One of these theorists, Cattell (1967), refers to fluid intelligence, g(f), and crystallized intelligence, g(c).

Cattell conceptualizes fluid intelligence as an ability to perceive relationships in material of many types and considers that it shows up best in areas requiring speed and in novel situations where new learning has to occur. He regards his own I.P.A.T. Culture-Fair Intelligence scales and the Progressive Matrices as good measures of g(f). Cattell argues that g(f) appears as a general factor because it is an underlying, unitary type of mental capacity or energy that is thrown from one type of performance to another. In other words, performances in different areas correlate with oneanother because the same force is exerted in different areas. In describing this concept, Cattell has used the analogy of different grades of petrol being employed in a variety of engines. If a higher grade

of petrol is used, it boosts performance in all engines. Research using tests that are taken to index this form of intelligence, suggests that it reaches its peak at about the time brain growth stops - i.e., at age 14 to 16. It then levels out and starts to drop from about age 22. Although failure to correct for generation effects in the earlier cross sectional studies has been shown by recent longitudinal investigations to exaggerate the rate of this decline, the generalization still holds.

Crystallized intelligence is regarded as a broad factor in an array of crystallized abilities that is built up through the interaction between fluid intelligence and experience or learning. In Cattell's (1967) words:

> "...crystallized intelligence is a collection of skilled judgements a person has aquired by applying his fluid intelligence to his school opportunities. It is a sort of 'holding company' for what fluid intelligence and school experience have jointly produced, and as such, it has a life of its own in that its skills tend to generate more skills like them". (p. 304)

Cattell regards skills like vocabulary level, arithmetic, and comprehension to be good measures of g(c). In contrast to tests presumed to tap g(f), these measures of intellectual functioning show continued development past the teenage years, flatten out in middle adulthood, and decline little with age until approximately five years before death. In other words, g(c) tends to retain the 'shape' that fluid ability and experience

have given it.

The relevance of this discussion to the topic of the present section was implied earlier when it was noted that alcoholic samples tend to do worse on the performance subtests of the Wechsler-Bellevue and the WAIS than do matched non-alcoholic controls. Although these empirically derived clinical tests were not developed with the fluid-crystallized concept in mind, it would seem that the performance tests reflect relatively more of g(f) than the verbal tests, tests which in the case of Vocabulary and Information, are the two indices that consistently "retain their shape" in alcoholic populations and are presumably indicative of overlearned or "crystallized" abilities.

Further studies add weight to the view that fluid intelligence might be impaired in many alcoholics. For example, Jones (1971) found that on Raven's Progressive Matrices (a test Cattell regards as a good measure of g(f)), alcoholics were moderately impaired. In addition, degree of impairment increased with number of years of alcoholic drinking.

Hebb has also developed a bipartite conceptualization of intelligence similar to Cattell's. However, as with Cattell's framework, it has not been applied directly to the study of intellectual functioning in alcoholics. In contrast, Halstead's (1947) and Reitan's (1955)

theories, and the tests that developed from them, have been applied in this area. These writers make a distinction between psychometric or general intelligence and biological intelligence. Psychometric intelligence is a similar concept to crystallized intelligence. Tests measuring this construct are considered to be largely verbal in content, strongly related to past learning, and are primarily measures of long-term memory. In contrast, biological intelligence bears a similarity to fluid intelligence. Tests which are regarded as indices of this trait are largely nonverbal and cannot be handled by recourse to long-term memory and past learning.

The Halstead-Reitan Battery, which was designed to operationalize the construct of biological intelligence, includes a number of novel problems where the relevant information for each task's solution is generally provided in the test setting. Goldstein (1976) claims that performance on these tasks is indicative of an individual's level of "complex adaptive ability", a group of high level skills including planning, foresight, and decision-making.

Overall performance on the Halstead-Reitan Battery is claimed by its authors to reflect the biological integrity of the brain. A well-established neuro-psychological literature supports this claim and shows that the battery is highly successful in both quantifying degree of organic involvement and locating specific sites

of cortical pathology (Golden, 1978). This aspect will be discussed in the next section.

On the Halstead-Reitan Battery, it has been demonstrated consistently that overall performances of alcoholic samples are sufficient to implicate neurological impairment. In terms of psychological functioning on this battery, Fitzhugh et al (1965) found that alcoholics show a severity of deficit that is more similar to acutely brain damaged populations than to non-alcoholics and normal controls. This study and its findings were replicated by Fitzhugh et al (1960). Considering the results of these studies in more detail, it is evident that the pattern of functioning found in the alcoholic groups were also qualitatively different from that of the nonalcoholic and brain damaged controls. Whereas the nonalcoholic controls failed to show impaired performance on any of the tests and the brain damaged group showed a relative deficit in all areas tested, the alcoholics were intermediate on all measures except the finger tapping test, a measure of motor skill on which they performed well, and the category test, where they performed at an inferior level to the brain damaged group. Independent investigations by Goldstein and Shelly (1971), Smith et al (1973), and Schau and O'Leary (1978), have corroborated these findings.

From this review, a consensus view seems to be emerging, strongly suggesting that after the withdrawal phase, during the first and second month of abstinence,

alcoholics have preserved "psychometric" or "crystallized" intelligence along with moderate to severely impaired abstract reasoning and ability to solve complex and novel problems. The overlearned, primarily verbal skills that rely on past experience and long-term memory, and that are indexed by the majority of the Wechsler-Bellevue and WAIS subtests, appear to hold up at previous levels. These are the types of skills that give the appearance of intactness in everyday activities. The apparent deficits in "biological intelligence" or "fluid intelligence" on the other hand, are more subtle, often only evidencing themselves in specific test situations. These deficits could be expected however, to have implications for behaviour in other non-test situations. From these considerations, it is evident that a "common sense" appraisal, or an assessment using a standard intelligence test, will give a very deceptive account of an alcoholic's intellectual capacity.

Before we leave this area of functioning, a cautionary note is in order. It is unclear from the literature to what extent the above generalizations apply. Discussion has been oriented around modal performance, playing down the considerable variance found in this area in alcoholic populations. As will be shown shortly, it is also unclear how permanent or reversible these deficits in intellectual capacities are.

Perception. Tarter (1976) has reviewed the literature

that addresses the perceptual capacities of alcoholics. The results of investigations in this area are mixed, showing impairments on some tasks but not on others. For example, perceptual speed appears to be intact. Perceptual-motor coordination, as measured by the Bender-Gestalt, on the other hand, has been shown to be impaired in some studies, whereas in others (e.g. Reinhr & Golightly, 1968) only a small percentage of the subjects evidenced signs of impairment. Other studies using different measures of perceptual-motor functioning (e.g. star tracing, Purdue Pegboard) have also yielded evidence of impairment. However, on the latter measure, there is some evidence of complete recovery after three months of abstinence (Tarter & Jones, 1971).

One point that is now fairly clear is that where perceptual-motor tasks rely heavily on spatial appreciation or ability to conceptualize spatial relations, impairment is found. Tests used in this group of studies include the Wechsler-Bellevue and WAIS Block Design subtest, the Grassi Block Substitution test, Kohs Block Design Test, and the Tactual Performance Test from the Halstead-Reitan Battery.

The most studied aspect of perceptual functioning in alcoholics has been perceptual field orientation or field dependence-independence as it is more commonly known. Performance on tests claimed to operationalize this construct is said to differentiate individuals in terms of their capacity to make accurate perceptual

judgements divorced from the contextual cues of the perceptual field. Many studies have found that as a group, both male and female alcoholics tend to perceive the world in a way that is strongly dominated by the surrounding field. In other words they rely more on the appearance of the environment than they do on internal cues or sources of information in making judgements. Studies based on the Rod and Frame Test (RFT) that have shown this enhanced field dependency in alcoholics are numerous. Examples include; Bailey et al, 1961; Donovan et al, 1976; Goldstein & Chotlos, 1965; Goldstein & Shelly, 1971; Jacobson, 1976(b);Witkin et al, 1959; Karp et al, 1963.

The field dependence-independence construct has been particularly fruitful in generating research across a broad front in experimental, personality, and clinical psychology. It is not intended to make a review of this literature here. However, it is relevant to this thesis to note that this literature has linked field dependence to a wide range of personality and cognitive processes. It has been argued, for example, that field dependent individuals also depend very heavily on the social context for self definition. Such individuals are also claimed to respond generally in a more global, less differentiated way, and to employ less sophisticated defence mechanisms such as denial and repression. In contrast, field independent individuals are considered to depend more upon intellectualization and isolation.

The presence of field dependence among alcoholics, at least as measured by the Rod and Frame Test, is now undisputed. There is considerable controversy however, with regard to the origin of field dependency in alcoholics. Taking the lead from Witkin et als' (1962) conclusion from the wider field dependence literature that performance on RFT is "..resistant to change by experimental means and ...appears to be a stable characteristic of the person" (p.373), it has been argued that field dependence is a stable characteristic of alcoholics throughout their lives. Further, it has been suggested that this trait in some way predisposes alcoholics to follow their particular 'career' path (Witkin et al, 1959; Karp et al, 1965a, 1965b; Karp & Konstadt, 1965). Although no longitudinal studies have yet been carried out to test this assertion, the studies by Karp and Witkin cited above, add some indirect support to this position. For example, they have shown that field dependence in alcoholics is not influenced by duration of drinking history, length of sobriety, or acute alcohol administration.

In recent years, the predisposition and stability arguments have been challenged. For example, in some populations, a wide heterogeneity of responses are evident, in some instances tending toward bimodal distributions (Goldstein & Chotlos, 1965). Other studies have demonstrated changes in mean field dependency scores towards field independence following a period of abstinence (Goldstein & Chotlos, 1966; Chess et al, 1971; McWilliams et al, 1975; Smith & Layden, 1972). On the

final test occasion in studies where RFT is repeated after varying periods of abstinence, the alcoholic group is still relatively field dependent however, albeit somewhat less so than on the first testing.

One may reasonably speculate that this decrease in field dependence over time in treatment was a result of detoxification. However, a similar magnitude of change on the RFT has been produced in a sample of alcoholics through exposure to one hour of sensory isolation (Jacobson, 1968). Jacobson considered that the change occurred because this experience focussed attention on to body sensations and internal cues as opposed to interfering external stimuli. Given this finding and possible explanation, it is of interest that the practice of meditation techniques, procedures which also involve a period of reduced external stimulation and increased awareness of internal stimuli, have been shown to produce increased field independence on the RFT in non-alcoholic subjects (Linden, 1973; Pelletier, 1974). These results, coupled with Chess et als' (1971) finding that RFT changes were accompanied by an increase in internality on Rotter's (1966) Locus of Control Scale, suggest that rather than a consequence of detoxification, increased field independence may be linked with a change in awareness and/or feelings of control over the body and self. Exposure to psychological therapies and the provision of time for reflection on past and present experience and feelings may also contribute to an increased focussing on the self that could conceivably relate to

this movement away from extreme field dependence.

Some researchers have suggested that RFT field dependence in alcoholics may be of a special type, possibly unrelated to developmentally acquired field dependence. However, this view has not been supported. For example, Goldstein et al (1970), employing RFT and a variety of other measures that are related to this test, failed to find differences between field independent alcoholics and nonalcoholics or between field dependent alcoholics and nonalcoholics. These results suggest alcoholics are not field dependent for some reason unique to them as a group.

Related to the view that field dependent alcoholics represent a special case, is the hypothesis that alcoholics are field dependent because of a more general acquired cognitive dysfunction and/or brain damage. In other words, their field dependence may relate to deficits such as those already noted in relation to intellectual capacity. In this respect, it is worth noting that diffusely brain damaged patients are the only diagnostic group in which greater field dependency has been observed.

If RFT field dependence in alcoholics is due to brain damage however, it should correlate significantly with measures that are known to reflect the organic integrity of the brain, i.e. neuropsychological tests. To test this possibility, Goldstein and Shelly (1971) administered RFT to a group of alcoholics, along with

the WAIS, the full Halstead-Reitan test battery, and the Trail Making Test. From a factor analysis, five factors were extracted. RFT loaded on only one factor, a factor on which only two other tests loaded, the WAIS Object Assembly Subtest, and the Trail Making Test. However, RFT failed to correlate significantly with the vast majority of tests known to reflect cognitive dysfunction and organicity in alcoholics. Another study using alcoholic subjects (Pisani et al, 1973) found no relationship between cognitive dysfunction as measured by the Shipley Institute of Living Scale (Shipley, 1940) and RFT. In contrast to these findings, a study of RFT and Halstead Neuropsychological test performance in normals, indicated significant correlation between RFT and a wide range of neuropsychological tests (Neuringer et al, 1975). However, the relevance of this result is uncertain as the subjects were not alcoholics and the RFT score range did not extend into the extreme of field dependency found among alcoholics. Further, even in this study, it was noted by the authors that although significant, the relationship was only slight.

Thus, the claimed association between RFT and other forms of cognitive dysfunction in alcoholics is not well supported. Donovan et al (1976) conclude from a review of the relevant literature: "...RFT performance may measure a particular form of intellectual functioning having high loadings on visual perceptual abilities, but" ..."may be relatively insensitive to changes in mental status" (p.736).

(i.e. other types of cognitive or neuropsychological dysfunction).

In conclusion, from the review of the literature relating RFT performance to alcoholism, although field dependence in alcoholic populations is a well established finding, the research to date does little to clarify the issue of whether it is an antecedent or a consequent of alcoholism. Although changes toward field independence over time in treatment have been found in four studies, it is not known for certain what causes these shifts. The present writer is of the opinion that the shift is at least in part a function of experiences occurring during the treatment period rather than following from internal changes associated with 'drying out'. At the present time however, this proposition is best regarded as a speculation. Although there is possibly some link between RFT field dependence and some aspects of alcoholism related cognitive dysfunction, in the main the two areas of functioning appear to be orthogonal to each other.

A further factor that has provided an obfuscation to attempts to clarify the perceptual and cognitive processes that might underly field dependence in alcoholics and explain shifts over time, is the demonstration that the two most commonly used field dependency tests, the RFT and Embedded Figures (EF), appear to be operationalizing rather different constructs. Although they correlate significantly, the common variance shared by these measures is only about 20 percent (Adevai et al, 1968; Gross & Moore,

1970; Witkin et al, 1971). Although it is uncertain what accounts for the shared variance between these measures, a study by Goldstein et al (1970) suggests that it stems from certain components of spatial functioning that are measured by both tests.

Given the quite large degree of statistical alienation that exists between RFT and EF, it is of interest that unlike RFT, the latter test does not always differentiate normals from alcoholics (Tarter, 1976). Additionally, in alcoholic populations, they relate in different ways to cognitive and personality measures. Whereas 'field dependence' measured by RFT appears to be largely independent of most other measures of cognitive dysfunction and covaries with variables that are typically viewed as indices of personality, e.g. locus of control (Chess et al, 1971) and inner directedness (McWilliams et al, 1975), EF performance covaries with degree of performance decrement on a wide variety of tasks shown to be particularly sensitive to cognitive deterioration in alcoholics (Donovan et al, 1976; O'Leary et al, 1977).

Like RFT, EF performance has also been shown to move in the direction of decreased 'field dependence' over time in abstinent alcoholics (O'Leary et al, 1977). However, from a consideration of the relation of EF to other cognitive measures, it seems reasonable to postulate that whereas shifts in RFT may be due to increased awareness of bodily cues and sensations, EF

shifts are likely to result from improvements in cognitive processes such as abstract reasoning, perceptual-motor functioning, and short-term ('secondary') visual-spatial memory. From these considerations, it does not appear to be justified to regard these two tests as measures of the same psychological function - an assumption that has been widely made in the literature to date.

From the field dependence literature and other studies of perceptual functioning in alcoholics, it seems reasonable to conclude that some degree of perceptual impairment exists among many abstinent alcoholics, particularly in the perceptuospatial area. However, not all aspects of perception are impaired and it is uncertain how reversible some of the observed deficits are. Certainly some show some recovery with continued abstinence. Although most of the deficits follow from prolonged alcohol abuse, at least in one area, field dependence as measured by RFT, there is some evidence consistent with the view that at least some, if not the majority of this impairment, antedates alcoholic involvement.

Motor functioning. From a review of the relevant literature up until 1976, Tarter (1976) concluded that there is strong evidence for muscular and motor disturbance in chronic alcoholics but, that whether or not such deficits are detected depends to a large degree upon the particular test and the task demands. These findings

are not unexpected given the demonstration of peripheral neuropathy, reduced nerve conduction velocities, decreased amplitude of neuronal action potentials, and muscle atrophy, among large numbers of alcoholics. Although these anatomical, physiological, and behavioural impairments to the motor system have been shown to be relatively common, clinically obvious peripheral neuropathy and myopathy occurs in less than five percent of alcoholics in treatment settings. This parallels the situation already discussed in relation to the Central Nervous System, where five to ten percent of unselected patients evidenced obvious clinical signs of an organic brain syndrome, but where more detailed neurological and psychological investigations implicated subclinical deterioration in somewhere over 50 percent.

Memory. From experience interviewing and assessing alcoholics within the first few weeks of admission to treatment programmes, the author has been impressed by the frequency of complaints made by patients regarding their memory capacity. Other clinicians have also found this (e.g. Parsons & Prigatano, 1977). Difficulties are often claimed in ability to recall names, appointment times, lecture material, and the location of programme activities. Severe memory deficits are one of the primary diagnostic criteria in the alcoholic brain syndromes. Among Korsakoff alcoholics, chronic memory disorders have been extensively investigated (see Butters & Cermak, 1976 for a recent review). Some of this work was described earlier in this chapter. It was noted

that there appeared to be a parallel between certain of the deficits in the Korsakoffs' secondary memory processes and deficits found in acutely intoxicated nonalcoholics. It was also noted that these areas of research are starting to draw on the conceptual framework of cognitive psychology to guide experimental programmes and assist in the interpretation of current findings. Research has also recently addressed the memory disorder known as the 'blackout phenomenon' - the inability of an individual while sober to recall events that took place during a previous period of intoxication (Goodwin et al, 1969, 1970, 1973).

It is curious, given the clinical observations and accounts by alcoholics themselves of memory disturbances while sober, the very active experimentation into aspects of memory functioning in the acutely intoxicated and the chronic disorders of Korsakoffs, that there is a dearth of literature relevant to the question of memory impairment in abstinent alcoholics. It is even more curious given the finding from animal experimentation, that the long-term ingestion of ethanol produces chronic decrements in memory and learning (Freund, 1973, 1975). In the main, the few relevant studies have been piecemeal and atheoretical in their conception. They have failed to consider theoretical models from cognitive psychology and normal memory processes, and have relied more upon empirically derived clinical instruments, instruments developed initially to aid in diagnostic decision making.

One commonly used clinical test of 'global' memory functioning, the Wechsler Memory Scale (Wechsler, 1945), has been used with alcoholics. Parsons and Prigatano (1977) administered this test to four matched groups of patients: alcoholics with a self-reported history of head injury; alcoholics without head injury; nonalcoholic head injury patients; and psychiatric patients (various diagnoses excluding brain damage and alcoholism). Alcoholic subjects were tested in the fourth and fifth week of treatment. On overall performance, only the nonalcoholic head injury group differed significantly from the other groups and showed evidence of memory impairment. In other words, on this measure alcoholics, irrespective of whether or not they reported previous head injury, performed at the appropriate level of memory functioning for their intellectual level.

Although the alcoholic subjects in Parsons and Prigatanos' study did not evidence overall memory impairment, they did show deficits on two of the subtests of the Wechsler battery. On the Associate Learning Subtest, alcoholics performed significantly worse than the nonalcoholic psychiatric control group. The other subtest to reveal impairment in the alcoholics relative to the psychiatric group was the digits reversed section of the Digit Span Subtest.

Claeson and Carlsson (1970) also noted a moderate learning deficit among alcoholics on a paired associate task. However, on a similar paired associate task,

Jonsson et al (1962) failed to find this type of deficit. They did however, observe relative impairment in the ability of alcoholics to remember a display of common objects. As is so often the case in this field, it is difficult to know what to make of the discrepant findings on paired associate learning tasks because the alcoholic samples were not sufficiently described to allow meaningful comparison across studies. As far as verbal memory is concerned, it seems from the studies to date that after the first two or three weeks of abstinence, if memory deficits are present, they are subtle. It may be that more refined tests, designed to operationalize some of the concepts that cognitive psychologists have applied to normal memory mechanisms (for example, the "levels of processing" model) may be more illuminating. Experiments described on page 46 were cast in this framework. Although the focus was on memory deficits in Korsakoff patients, the alcoholic control groups in some of these studies performed at a level intermediate between that of the normal controls and the Korsakoffs. These findings suggest that this approach may be more productive in identifying subtle memory deficits in sober alcoholics.

Although the evidence for verbal memory deficits in alcoholics is equivocal, there is some research suggesting that disturbances in nonverbal memory may be present. For example, Claeson and Carlsson (1970) found that 65 percent of an alcoholic sample performed in the impaired range on the Benton Visual Retention Test. In contrast to this finding however, May et al

(1970) failed to find impairment on the related Memory for Designs Test. Although alcoholics recall tactually perceived forms (as measured by the Tactual Form Board of the Halstead Battery) as well as controls (Fitzhugh et al, 1960, 1965), they are impaired in their ability to locate forms on the form-board (Prigatano, 1977). Parsons and Prigatano (1977) consider that this finding indicates an impairment in recall for spatial location.

In summary, there is no firm evidence of 'global' or general memory impairments in abstinent alcoholics. There is however, some experimental corroboration for the view that more subtle deterioration is present. In particular, certain aspects of verbal memory, perhaps ability to spontaneously encode or to efficiently employ semantic processing strategies, and some types of nonverbal, particularly spatial memory, seem to be impaired. In conclusion however, in spite of the strong suggestion of deterioration from clinical observation, and the obvious importance that such deterioration could be expected to have for treatment participation and perhaps treatment outcome, very little can be said with any certainty regarding the presence, kind, extent, and duration of memory disturbance of sober alcoholics. Although in part this state of affairs is a consequence of inadequate conceptualization, poor methodology, and deficient descriptions of the alcoholic samples used, it is primarily due to a lack of research interest in this area.

Abstraction. It was noted earlier in this chapter that on the Halstead Reitan Battery, alcoholics show a mean performance level intermediate between brain damaged patients and matched nonalcoholics (Fitzhugh et al, 1960, 1965; Goldstein & Shelly, 1971; Jones & Parsons, 1971). Although this generalization applies to most of the Halstead-Reitan subtests, it was pointed out that on one of these measures, the Category Test, alcoholics did very poorly. Indeed, they performed at a level below that of groups with documented brain damage. This task requires subjects to learn general principles that relate sets of geometric forms to each other. The capacity to perform this type of task has been referred to as 'abstract attitude' by Goldstein and Scheerer (1941). Abstract attitude describes the ability to generate a principle or concept that links entities that have some attributes in common but differ in many ways.

Studies using the Halstead Category Task and the Wisconsin Card Sorting Task, a related abstraction test, have consistently corroborated the view that alcoholics are severely deficient in abstraction ability. They have also shown that, in contrast to many brain damaged patients who have difficulty aquiring concepts, alcoholics typically display a particular type of abstraction deficit. More specifically, rather than showing deficits in ability to aquire concepts or by making perseverative errors, alcoholics evidence severe difficulties in flexibility (shifting from one concept to another) and inadequate persistence in sticking to new strategies. It has also

been argued that these abstraction deficits in alcoholics are confined to one modality, that involving visual-spatial stimuli. That is to say, it is not a generalized deficit. Experimental data supporting these generalizations have been collected in Goldstein's (1976) and Tarter's (1976) reviews. Further findings of interest here include the demonstration that flexibility deficits are largely confined to alcoholics with a history of over ten years of alcoholic drinking. Persistence deficits on the other hand, are more apparent in longer term alcoholics but, also occur in short-term alcoholics (Tarter, 1971; Tarter & Parsons, 1971).

Other tests of 'abstract attitude' or abstracting ability have also revealed impairments in alcoholics relative to nonalcoholic controls. Examples include proverbs tests (Jonsson et al, 1962) and a modified form of the Goldstein Object Sorting Test (Lovibond & Holloway, 1968). Some studies using measures claiming to tap this area of functioning have failed to demonstrate impairment (e.g. Claeson & Carlsson, 1970; Pishkin et al, 1972; Tarter et al, 1975).

Thus, as in the other areas of psychological functioning reviewed here, general impairments have not been demonstrated in all areas of abstracting ability. However, selective impairments have been consistently documented, particularly in persistence and flexibility with regard to concept-formation tasks using visual-spatial stimuli.

Conclusions. The literature reviewed in this section indicates that on tests measuring over-learned, predominantly verbal skills and knowledge accumulated over many years, alcoholics perform at a similar level to nonalcoholics of the same age. For this reason, overall I.Q. scores that depend heavily on verbal facility and long-term learning, fail to discriminate between abstinent alcoholics and nonalcoholics living in the community. Facility in these areas gives an appearance, in most day-to-day situations, of an adequate level of functioning.

In contrast to this pattern of well-preserved "crystallized intelligence", alcoholics have considerable difficulty solving certain types of novel problems, particularly problems involving the manipulation of abstract concepts. In addition to specific abstraction deficits, alcoholics as a group perform poorly on a variety of complex psychomotor tasks, especially when these tasks have a spatial component. Indeed, in all of the areas of psychological functioning discussed (intelligence, perception, motor performance, memory, and abstraction), spatial deficits, particularly in visual-spatial performance and spatial location skill, were implicated in the patterns of impairment observed. There is also the suggestion that some aspects of verbal secondary memory are moderately impaired.

Most if not all of the above deficits appear to be in the general area of cognitive or intellectual

functioning that has been referred to variously as fluid intelligence and biological intelligence. Unlike the areas where alcoholics display intact performance, deficits in this area are not obvious in daily routine activities. However, they have been claimed to be particularly relevant to ability to make behavioural adjustments to new situations as well as to planning and decision-making that involves the integration of new information or conceptualization of the self in relation to future events or situations.

Although some of these psychological deficits are considered to antedate a history of alcohol abuse, the strongest case for this being with regard to field dependence as measured by the RFT, most of the available evidence suggests that these changes occur as a consequence of alcohol abuse. Some corroboration for this view comes from studies already cited, where correlations between years of problem drinking and severity of behavioural deficits were found. Further inferential support comes from the demonstration that some of the behavioural deficits are either partly or totally reversible after weeks or months of abstinence.

Because of the apparent reversibility mentioned in the last paragraph, the cognitive impairment of alcoholics differs from patient groups suffering from miscellaneous forms of brain damage. The specificity of the areas of dysfunction are also distinctive to the chronic alcoholic, although they show some continuity with both

of the two most common alcohol-related organic brain syndromes. In some areas they also show a parallel with the effects of acute intoxication upon nonalcoholics. The suggestion that such short-term acute changes can in time become reflected in less readily reversible changes in cognitive functioning, also corroborates the view that alcohol produces the pattern of dysfunction found, rather than the pattern producing the problem with alcohol.

Although the literature allows some tentative conclusions to be reached, there are still many questions that have yet to be answered regarding intellectual and cognitive functioning in alcoholics. And, more generally, we know little of how alcoholics function cognitively outside of testing situations embedded within treatment programmes.

Neuropsychological Models

Neuropsychological tests are psychological tests that have been designed to be selective to the biological condition of the brain rather than to measure or to delineate psychological processes per se. Neuropsychology has developed very rapidly in the last ten years. It is concerned with the development of psychological measures that aid in the diagnosis and location of brain injury, with untangling the relationship of psychological functions to specific structures and pathways in the brain, and is now starting to ascertain the relevance of this accumulating body of knowledge to the design of

rehabilitation programmes for brain damaged patients (see Golden, 1978 for an overview of this field).

The performance of alcoholics on a variety of neuropsychological tests has been described in the previous section, where the major concern was the description of certain psychological capacities of sober alcoholics. Because the performance of alcoholics on some of these measures resembles performance associated with certain types of brain damage, it has been argued (e.g. Tarter, 1975, 1976) that neuropathological changes are responsible for the behavioural findings. In this section, additional neuropsychological findings will be linked with the studies outlined in the previous section and organized around the major neuropsychological models that have been proposed to account for cognitive dysfunction in alcoholics.

Premature aging hypothesis.

It has been argued that alcohol abuse leads to a premature aging of the brain and that this in turn produces the behavioural deficits noted in many alcoholics. Anatomical and neurological data are available that suggest parallels between normal aging and the effects of excessive alcohol intake. Courville (1955) for example, considered that the brains of alcoholics at autopsy resembled brains of the very elderly. Smith et al (1971) found that sleeping EEGs of alcoholics were similar to recordings taken from elderly persons.

Neuropsychological studies also suggest a parallel between the effects of normal aging and alcoholism. Korboot and Naylor (1972) concluded that prolonged alcohol abuse produces a variety of mental dysfunction, analogous to that resulting from the normal process of aging. From WAIS scores and a measure of information acceptance rate, they concluded that the performance of their alcoholic sample closely resembled that of normal elderly persons, more than 20 years their senior. In a subsequent study, using alcoholics with a briefer alcoholic history, they found a similar qualitative pattern of dysfunction, although quantitatively it was intermediate between nonalcoholic social drinkers of the same age and the alcoholics in their earlier study (Korboot et al, 1977). Again they noted a resemblance between the score patterns of alcoholics and the aged.

Fitzhugh et al (1965) regarded the Halstead-Reitan performance of alcoholics as an indication of premature aging in terms of adaptive abilities or biological intelligence. Earlier, it was suggested that biological intelligence and fluid intelligence are virtually synonymous terms. A well established finding in the investigation of the psychological effects of aging is that many of the tests claimed to reflect fluid intelligence are particularly sensitive to the effects of aging. Measures that are regarded as indices of crystallized intelligence on the other hand, show little deterioration with advancing chronological age. Here there

is an obvious parallel with the overall impression reached from the earlier review of the types of intellectual deficits found among alcoholics. In other words, like elderly people, many alcoholics obtain lower scores on tests that require mental flexibility and perceptual-motor speed relative to those that tap established or practiced cognitive abilities.

Although the analogy between alcoholism and premature aging appears to have some validity, it may be oversimplistic. For example, Overall and Gorham (1972) noted that there are two types of age-related deterioration. One, which is seen as part of the normal aging process, involves a decline in those tasks considered to reflect fluid intelligence. Also relatively common among the elderly, although by no means representative of all aged individuals, is a more general behavioural impairment which is considered to be a pathological condition. On the WAIS, this leads to a depression of both Verbal and Performance Subtests, in contrast with normal aging which produces an irregular "saw tooth" pattern. Individuals with generalized deterioration typically have a chronic brain syndrome which involves diffuse cortical deterioration. Based on a study of WAIS subtest scores in old people with chronic brain syndromes (with acute and chronic focal lesions excluded) and normal elderly subjects, Overall and Gorham derived discriminant functions that maximally separated the two groups. Williams et al (1973) subsequently used these WAIS derived aging and organicity

functions to determine whether the preclinical changes associated with alcohol abuse are more consistent with accelerated mental aging or a developing organic brain syndrome. The WAIS subtest profiles produced by their hospitalized alcoholic sample were similar to those found in other alcoholic groups (e.g. Matarazzo, 1972). They also demonstrated that their alcoholic sample deviated from age norms along both the mental aging and organicity dimensions. This finding, of course, conflicts with the view that the intellectual changes observed in alcoholics reflect only premature aging. On the other hand, it does suggest that some of the deficits observed in alcoholics can be regarded in this way.

There are also other difficulties in the premature aging hypothesis. One is that the analogy rests upon the assumption that the brains of the two groups are in a similar biological condition. As it happens, there is evidence that a variety of non-organically based factors play some part in producing cognitive changes in the aged. Sociocultural factors, communication capacity, motivation, and affective state (e.g. the common occurence of clinical and subclinical depression) are just some of the factors that affect test performance in elderly groups (Goldstein, 1976).

A related problem that produces some difficulties for the premature aging analogy has already been mentioned. A number of investigators (e.g. Schaie & Strother, 1968)

have demonstrated that longitudinal studies of cognitive functioning show a different picture from the cross-sectional investigations that dominated the early literature and provided the data base for the so-called classical curve of mental deterioration. This is a complicated issue. Although possibly an oversimplification, it now looks as if much of the difference in performance between age groups stems from differences in educational and other life experiences. When these factors are controlled in longitudinal designs, the deterioration in fluid intelligence is much more gradual, at least until shortly before death. Where the performance of alcoholics has been compared with that of the elderly, the elderly group has always come from an earlier generation. Thus, much of the similarity between the two groups probably arises from cultural factors rather than common biological changes. Consequently, although there are some commonalities between alcoholics and the elderly, the analogy cannot be pushed too far without distorting what is known of both groups.

Nonspecific cortical deterioration

Until recently it was assumed that the major organic consequence of prolonged alcohol abuse was diffuse CNS atrophy (e.g. Brosin, 1967). Pathology of this type is associated with generalized cognitive deterioration (senility). This type of deterioration is known to occur in a subgroup of the very elderly and in a small

percentage of advanced alcoholics (those with alcoholic dementia). However, it has been shown in the literature reviewed in this chapter that the majority of alcoholics do not typically display this form of generalized cognitive deterioration. To the contrary, with the exception of Williams et als' (1973) study,suggesting that a part of the cognitive decline of alcoholics can be accounted for by movement along this dimension, the weight of evidence points to deficits in specific intellectual and cognitive tasks. Similarly, the anatomical and neurological studies reviewed indicate that a major feature of alcoholism is the presence of localized atrophy. However, post mortem studies also show that a localized focus of atrophy appears to be superimposed upon diffuse pathology reflected in both the macrostructure and microstructure of the cortex. Because diffuse deterioration is less readily detectable in neurological examinations, it remains uncertain to what extent diffuse changes are present in the earlier stages of the alcoholic deterioration process. If they are present at this point, the question remains as to why they do not appear to express themselves more clearly in behavioural changes.

Right hemisphere dysfunction

Neuropsychological research has established that the human brain is organized into relatively well defined areas which mediate different kinds of psychological functions (see Dimond & Beaumont, 1974; Luria, 1973,

Golden, 1978). Within the cortex, it has been shown that in most individuals, the left hemisphere serves the functions of language, symbolic processes, complex motor organization, high level vigilance performance, associative learning, and control of the right side of the body. The right hemisphere is much less well organized functionally, but is believed to be the primary site for spatial integration, calculation, and creative, associative thinking.

It seems that most forms of so-called 'higher' mental activity require the integrated workings of the entire intact brain for optimal functioning. Nevertheless, the pattern of strengths and deficits among these functions can provide information regarding the severity of brain damage, the lateralization of the injury, and even its more specific locus within a hemisphere.

From the pattern of psychological deficits reviewed earlier, a number of writers (e.g. Parsons et al, 1972), have argued that the right hemisphere is more prone to damage from chronic alcohol abuse than is the left hemisphere. The findings from studies of intellectual performance among alcoholics form one body of research that has been claimed to support this position. For example, it was noted previously that on the Wechsler scales, alcoholics typically obtain significantly poorer performance than verbal IQs. In clinical lore, this discrepancy has long been regarded as diagnostic of right as opposed to left hemisphere dysfunction. Recently

however, empirical investigations have challenged the validity of this claim. For example, Todd et al (1977) found that the verbal-performance discrepancy failed to provide any discrimination between left, right, diffuse, and nonspecific brain damaged groups.

Although it can no longer be maintained that the lower performance IQs of many alcoholics unequivocally corroborate claims for right hemisphere dysfunction, other aspects of alcoholics' intellectual functioning fare better in this respect. This is because although the more gross IQ scores are not particularly sensitive to lateralized organic deterioration, individual subtest performances are (McFie, 1975; Swiercininsky, 1978).

It was shown in an earlier section of this chapter (page 49), that impaired performance is typical among alcoholics on the Digit Symbol, Object Assembly, and Block Design Subtests of the WAIS and Wechsler Bellevue. Digit Symbol performance is of little value to the question of lateralization. This is because the task is sensitive to disruption in a very wide variety of psychological functions and appears to be equally reflective of lesions in most, if not all, parts of the cerebrum (McFie, 1975). Object Assembly and Block Design on the other hand, are both relatively pure tests of visual-spatial organization. In normal adults, visual-spatial analysis and performance are mediated by the right hemisphere, and impairment on these tasks when other subtest scores are relatively intact, has been

strongly associated with the presence of right hemisphere pathology, particularly in the right parietal area. Poor performance relative to controls on Raven's Progressive Matrices, a visual-perceptual intelligence measure, also corroborates this view. So too does the consistent finding of intact functioning on predominantly left hemisphere mediated verbal intelligence measures such as the WAIS Vocabulary and Information Subtests and the Shipley-Hartford Scale.

Earlier it was argued that in those psychological processes (intelligence, perception, motor performance, memory, and abstraction) where deficits have been demonstrated in alcoholics, deterioration in aspects of spatial ability was apparent. Indeed, it could be the major deficit underlying deterioration in these various functional areas. However, again as noted earlier, not all spatial tasks are found to be disrupted in alcoholics.

Other work consistent with the right hemisphere hypothesis includes the finding of impaired motor control and inhibition in the left hand of alcoholics relative to nonalcoholic controls, while both did equally well with their right hand (Parsons, 1975). This fits in with Vande Vusse's (1976) observation that left hand performance on the Purdue Pegboard was correlated with years of alcoholic drinking, but right hand performance was not. On dichotic listening tasks, alcoholics are more impaired on items delivered to the left ear (Goodglass & Peck,

1972). Because of the contralateral representation of sensory and motor functions, these studies suggest greater disruption of right hemisphere structures.

Two recent experiments were designed to directly address the right hemisphere hypothesis. Bertera and Parsons (1978) found that as predicted, long-term alcoholics took significantly longer than controls to process computer-generated nonverbal shapes. Also predicted from the lateralization hypothesis was the finding of no difference between the two groups when verbal stimuli (consonant-vowel-consonant trigrams) were used. Cutting (1978a) tested alcoholics after they had been abstinent for four weeks. He found significant differences between alcoholics and controls on a picture memory task (considered to be sensitive to right hemisphere dysfunction) and a verbal fluency task (regarded as an index of frontal lobe dysfunction). Alcoholics with heavy alcohol intakes also performed significantly worse on these two measures than alcoholics who drank less. Although alcoholics were deficient on a verbal paired associate learning task, the comparison with the control group failed to reach the .05 significance level. Two measures of abstract thinking (the Similarities Subtest of the WAIS and a proverb interpretation task) did not differentiate the groups. Cutting considered that the nonverbal memory deficit was probably a consequence of right temporal lobe dysfunction.

From the studies reviewed, the available evidence

suggests that chronic alcohol abuse has a greater effect on the right hemisphere than it does on the left. However, some of the findings do not fit this model. For example, the verbal fluency deficit noted in Cutting's (1978a) study suggests left frontal dysfunction (McFie, 1975). Other studies show impaired verbal short-term memory, particularly during the first two weeks of abstinence (Goldman & Rosenbaum, 1977). A variety of abstraction deficits have also been demonstrated, although admittedly, they do appear to be primarily of a spatial or nonverbal type. Psychological dysfunction in these non-spatial areas is considered to implicate brain structures other than the right hemisphere.

Tarter (1976) has made the point that because tests known to reflect right hemisphere functioning are particularly sensitive to disruption by alcohol, both when it is administered acutely and from long-term abuse, this does not necessarily mean that only the right hemisphere is impaired. Apart from the finding of deficits implicating other parts of the brain, it may be that right hemisphere lesions and other forms of biological insult are more readily expressed in psychometric performance. To put it another way, right hemisphere dysfunction might be easier to detect than left hemisphere dysfunction.

Other problems for the right hemisphere hypothesis arise from consideration of the senile dementias of

advanced alcoholics and the anatomical and neurological data reviewed earlier.

Alcoholic dementia has an insidious onset and is associated with many years of alcoholic drinking. Anatomically and psychometrically, this condition yields evidence of association with widespread diffuse cortical atrophy, to both hemispheres. Now, from whence does this syndrome spring? Earlier it was argued that there is some evidence for regarding alcoholic dementia as the extreme point on a continuum of non-Korsakoff alcoholic deterioration. If this is so, why should left hemisphere atrophy suddenly appear late in the day of the alcoholic's life? It would seem more reasonable to assume that rather than a late development with an acute onset, that both left and right cortical deterioration is present in a less advanced form earlier, but that left hemisphere tissue pathology only becomes obvious, psychologically, at more advanced stages. The anatomical and neurological investigations of less psychologically impaired alcoholics also support this view. The pattern revealed is one of diffuse deterioration, albeit usually with more severe atrophy in frontal and anterior subcortical, and to a lesser degree, parietal regions. These studies do not implicate differential right hemisphere pathology.

In conclusion, the alcoholic psychometric picture is one of predominantly right hemisphere involvement. However, rather than following from more advanced organic deterioration in this region, it more probably occurs

because the visuo-spatial systems of the right hemisphere are more vulnerable to the effects of alcohol-associated cortical disruption than are left hemisphere mediated overlearned verbal skills. Semmes (1968) has proposed a model for hemisphere specialization that provides an explanation for why this differential sensitivity to disruption could be expected to occur.

Anterior-basal dysfunction

Of the neuropsychological models of cognitive dysfunction, the view that alcoholics are neurologically impaired in frontal-limbic areas has received most attention in recent years. Because this region of the brain is complex, both from a neuroanatomical and a psychological point of view, a description of this area will be given before the relevant data from studies of alcoholics is discussed.

The large size of the frontal lobes, relative to other structures within the brain, is a major feature differentiating the human brain from the brains of other primate species. Not surprisingly, it is linked with many behaviours that are considered to be peculiarly human. The mediation of these behaviours rests upon the functioning of a number of interlocking systems which, to some extent, are characterized by specific localization within the frontal cortex.

The most posterior region of frontal cortex is

primarily a motor area, concerned with the execution of all voluntary motor activity, including speech. Adjacent to this is a secondary motor area which is responsible for integrating motor functioning into smooth, regulated behavioural sequences. In these two areas, the right cortex controls motor activities of the left side of the body and vice versa. Even relatively small lesions in these areas lead to major disruptions in motor activity and/or speech.

Anterior to the motor cortex is the prefrontal cortex or, following Luria's terminology, the frontal tertiary area. The lateral portion of this region is responsible for the anticipation of future events and for planning, structuring, and evaluating voluntary behaviour. Although lesions to this area do not leave people unable to anticipate the course of future events, it has been shown that such individuals are severely impaired in their ability to picture themselves in relation to such events as a potential agent. Typically, they are easily distracted and have difficulty sustaining attention during an ongoing activity. People so afflicted are also frequently very inflexible, finding it extremely difficult to change activities or to do things, either conceptually or behaviourally, in alternate ways.

As in the motor cortex, there is some hemispheric division of labour within this area. Large lesions in the left tertiary area can lead to difficulties with speech including less overt changes relating to behavioural

regulation. For example, an individual with a lesion in this area may propose to do one thing, and inform others of this intention, and then find him or herself doing something quite different. Related to this, and also common, is difficulty in translating verbal instructions into actions, particularly when the instructions are complex or symbolic. People with lesions in this area also have trouble integrating data from varied sources and, as a consequence, are prone to make decisions without gathering sufficient information. Deficits in verbal fluency are also common.

Left tertiary lesions have been associated with severely impaired performance on the following tests: Halstead Category Task; Wisconsin Card Sorting Test; Part B of the Trail Making Test; colour word page of the Stroop Colour and Word Test. These are all tests of categorization and flexibility. Deficits are also common on tests of word fluency and verbal associative learning. On the WAIS there is typically no impairment in overall IQ. Digit Span however, is often depressed, and when damage is extensive, there can be a large drop in performance versus verbal IQ. This drop occurs primarily because of impaired motor performance in the dominant hand. Less extensive lesions have also been linked with motor deficits. For example, low scores for dominant hand performance have been noted on the Finger Tapping, Purdue Pegboard, and Tactual Performance tests.

Large lesions in the right frontal tertiary cortex are associated with similar disorders to those described for the left, with the exception of speech and speech-related regulatory and abstraction deficits. Motor disturbance is relatively common, particularly when lesions extend to the posterior portion of this region, but less obvious in everyday behaviour because the hand involved is the nondominant one. Severe impairments are usually found in the areas of visual-spatial integration, maze learning, and nonverbal visual memory (Golden, 1978). As with nondominant motor functions, these behaviours are not obvious in most daily activities. Consequently, large lesions can occur in this area without obvious symptoms.

Neuropsychological testing with right lateral tertiary patients has revealed deficits on the Picture Arrangement and Digit Span Subtests of the WAIS with little or no change on overall IQ. Impaired performance has also been observed on the Memory For Designs test and on the Finger Tapping, Purdue Pegboard, and Tactual Performance tests when the nondominant hand was used.

Beneath the lateral tertiary region is another section of cortex, occupying the medial and orbital surface of the prefrontal lobes. Functionally, this area is linked with two important subcortical structures, the Limbic System and the Reticular Activating System. Unlike all other parts of the human cortex, there appear to be no reliable left-right differences in this

section of the prefrontal lobes. When injuries are localized to just this area, they are extremely difficult to detect behaviourally. This is because they generally result in emotional changes. Either no change, or very minor deficits are produced on neuropsychological measures of frontal integrity, for example, abstraction tasks (Golden, 1978). Typically, changes include lack of anxiety, reduced inhibition in social situations, impulsiveness, and either mild euphoria or apathy.

The Limbic System consists of a group of structures on and near the basal surface of the cerebral hemispheres. Morphologically, it is closely associated with the prefrontal lobes, certain portions of the temporal lobes, and the hypothalamus. Structurally and behaviourally, the area is extremely complex. However, looking at the functions of the area as a whole, they can be subsumed within four broad areas: memory, olfaction, autonomic visceral functions, and emotional behaviour (Moore, 1976). A number of neuroanatomists and neuropsychologists have argued that not only are prefrontal and limbic structures linked morphologically, but that they are functionally integrated in that they mediate common complex behaviours and psychological processes (Tarter, 1975). Although damage to different parts of this system can result in similar behavioural impairments, for example, in aspects of memory functioning and emotional changes (Brutkowski, 1965), from discussions to this point, it is also evident that there is also some functional division of labour.

Among alcoholics, those with Korsakoff's Psychosis show the clearest evidence of damage to the anterior-basal area, particularly the subcortical portion of this system. Studies show that individuals afflicted with this neurological condition have bilateral atrophy and pathological tissue change within the hippocampal gyrus and mammallary bodies. As in other clinical groups with damage to this area, the most marked behavioural sequala is a profound impairment within the secondary memory mechanisms for both verbal and spatial material. A more detailed account of the problems Korsakoffs face in processing and recalling verbal information was given earlier in this chapter. At that point, it was also noted that chronic alcoholics appear to have qualitatively similar, although much less severe, deficits in verbal learning. Post mortem studies and neurological investigations showing enlargements in the third ventrical, suggest that many alcoholics may show these deficits for the same reasons, neurologically, that Korsakoffs do. This is an interesting possibility because it has previously been thought that the Korsakoff condition, linked with an acute onset and thiamine deficiency, is a unique form of organic brain syndrome. To the contrary, it now appears as if chronic alcoholics evidence preclinical changes of this type.

One difficulty with arguments for neuropsychological continuity between Korsakoffs and other alcoholics lies with a small body of literature dealing with olfaction in the two groups. Important structures concerned with

the processing of olfactory stimuli are located within the limbic system. Although severe deficits have been demonstrated in the ability of Korsakoffs to detect, identify, discriminate between, and remember odours, alcoholics do not appear to show impairment in these capacities (Note 2). The question arises as to why the apparent pathology in alcoholics' short-term memory mechanisms does not extend to olfactory structures that are immediately adjacent? It may be that it does, but that olfactory mechanisms are more resistant to disruption. Alternatively, it could be that deterioration in the limbic olfactory structures does not account for the deficits noted in Korsakoffs. Work by Butters and Cermack (1976) suggests that the major locus for Korsakoff olfaction impairment may reside in specific thalamic nuclei rather than the limbic system. If this is so, this in turn raises the question of why these structures are not at least minimally disrupted in alcoholics when they are known to be damaged in Korsakoffs.

Earlier, a review of post mortem and neurological investigations indicated that the most common focus of brain atrophy in alcoholics was the frontal region. Widening of the frontal sulci indicates that this atrophy extends to lateral areas of the frontal cortex as well as to the medial and orbital areas. Tracts connecting the prefrontal lobes to the limbic system and hypothalamus were also shown to be disrupted in one of the studies. Electroencephalographic measurements have also implicated frontal and anterior-basal pathology (Brewer & Perrett,

1971; Lereboullet, 1956).

Given the strong evidence for anterior-basal atrophy from the neurological sciences, to what extent does this form of organic pathology fit in with the neuropsychological data?

When due allowance is made for the temporary neuromuscular pathology found among some alcoholics, there is no support for the view that gross motor or speech pathology persists in alcoholics. This suggests that anything other than very minor structural changes in the primary and secondary motor areas of the frontal cortex is unlikely. Nevertheless, literature has been reviewed that indicates disruption to motor tasks that require fine movements and speed. These studies also indicated that it was nondominant hand performance that was significantly deficient. Other studies (e.g. Vivian et al, 1973) have shown that motor performance is most impaired when the test situation requires the integration of perceptual information, particularly spatial stimuli, with motor output. All of these deficits are similar in nature to those associated with patients who have sustained damage to the lateral region of the right prefrontal lobe. Not consistent with damage to this area however, is the failure to demonstrate consistent deficits on either the Memory For Designs test or the Picture Arrangement Subtest of the WAIS. The depressed score on the Digit Span Subtest of the WAIS found in some studies is however in keeping with lesions

to both this area and to the corresponding left prefrontal region. But, not all studies including this measure have replicated this finding.

The most severe neuropsychological impairments found among alcoholics come from tests that are known to be particularly sensitive to organic pathology within the lateral area of the left prefrontal lobe. Indeed, some studies reviewed earlier in this chapter, found performance on some of these measures to be worse than that characteristic of demonstrably brain damaged groups. Some caution is necessary when interpreting these results however. For example, although the Halstead Category Task and the Trail Making Test have been shown to be particularly sensitive to prefrontal lesions and cognitive dysfunction characteristic of alcoholics, both of these tests are sensitive to brain injury in general (Golden, 1978). Nevertheless, the particular nature of the alcoholics' deficits on these tests, namely problems with flexibility and persistence, support the frontal damage hypothesis. Performance on the Wisconsin Card Sorting Test, indicating evidence of impaired set persistence, impaired set shifting, and failure to use errors as corrective information among alcoholics is also typical of the behaviour of patients with prefrontal lesions. Deficient performance among alcoholics on the Stroop Colour and Word Test, on tests of verbal fluency, and verbal associative learning also suggest a left frontal involvement. As already mentioned, the latter of these

deficits can apparently result from disruption of the prefrontal-limbic system at a number of points.

Also typical of patients with prefrontal damage, particulalry when it is bilateral, is an inability to temporally organize behaviour and an impairment of speed and accuracy in visual-spatial scanning (Luria 1966 (a), 1966 (b)). Deficits of these kinds have been demonstrated in alcoholics (Bertera & Parsons, 1978; Tarter, 1971, 1975). In one study, reduced visual search activity, particularly in the periphery of a visual array, was found during a driver simulation task. The alcoholics in this study had been abstinent for three months prior to testing (Beidman et al, 1976).

In contrast with left hand motor performance, deficits of a similar nature are not characteristic of the dominant hand. This finding is not in keeping with the results of the other studies that strongly suggest left lateral prefrontal dysfunction in sober alcoholics. The reason for this inconsistency is not known. It could be that the more posterior areas of the prefrontal cortex that are apparently most involved with this function, show greater resistance to behavioural impairment within the left hemisphere. It was argued previously that there was some support for this notion with respect to other posterior cortical structures.

Serial alternation tasks have been claimed to identify frontally impaired individuals (Luria, 1966a).

Only one study in the literature appears to have used this type of task with alcoholics (Tarter, 1971). In this study, the alcoholic subjects failed to display evidence of impairment. Tarter (1975) considers that the high field dependence scores typical of alcoholic populations supports the argument for frontal impairment in alcoholics. However, one wonders about the justification for linking field dependence to frontal lobe damage. High field dependence has been associated with brain damage, but the highest scores were from a group with diffuse cortical deterioration. In addition, it was noted earlier that performance on RFT was found to be orthogonal to performance on a wide variety of neuropsychological tests, a number of which were particularly sensitive to frontal integrity.

Anterior-basal injury, particularly when the orbital prefrontal area is involved, is related more to changes in personality and emotionality than to cognitive functioning. Increased extraversion is one personality change that has been documented in patients with frontal lobe injury (e.g. Meyer, 1961). Levinson and Meyer (1965) have also demonstrated large increases in extraversion and large decreases in neuroticism, as measured by the Maudsley Personality Inventory, among patients following modified leucotomy (orbital undercutting). These psychometric findings support clinical impressions of reduced social inhibition, impulsiveness, and reduced anxiety in these two patient groups.

Korsakoff alcoholics are noted for their flattened affective tone - often described as dullness, apathy, or lack of initiative (Pearce, 1977). These characteristics, and other personality sequelae of anterior-basal damage, have not been systematically investigated in alcoholics. Indeed, there appears to be only one study that has addressed this issue. Smart (1965), divided alcoholic subjects into 'brain damaged' (WAIS Verbal IQ at least 18 points higher than Performance IQ) and 'non-brain damaged' (Verbal-Performance discrepancy less than five IQ points) groups. The former group was found to have a significantly higher extraversion score on the Maudsley Personality Inventory and was considered by the author to indicate a personality change associated with brain damage. Although suggestive, this does not constitute firm evidence for frontally-mediated personality changes in alcoholics. Further work is clearly needed.

There is a very large literature concerned with personality functioning in hospitalized alcoholics. Many of these studies, particularly the earlier ones, have been preoccupied with the search for the alcoholic personality (see Neuringer & Clopton, 1976). Although these studies do not relate personality measures to indices of brain damage or cognitive functioning, they could be expected to have an indirect bearing on the issue of organically mediated personality changes in alcoholics. This possibility will be briefly explored shortly.

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Earlier it was noted that some of the most obvious effects of acute intoxication involve changes in emotionality and social functioning. Naturalistic observation and anecdotal reports suggest that decreased inhibition and anxiety are common. Given the parallels between other acute effects and more chronic changes in alcoholics, it seems reasonable to make similar extrapolations regarding emotionality and impulse control. Unfortunately, very little investigation of an empirical nature has been conducted on changes of this type during acute intoxication. Consequently, although plausible, the comments made here remain highly speculative.

Brain changes and neuropsychological deficits in alcoholics.

The studies reviewed in this chapter provide compelling evidence for the presence of both neurologically verified brain damage and neuropsychological impairment among hospitalized alcoholics. Although it is less certain what underlying mechanisms are responsible for the types of behavioural deficit observed, some fairly convincing parallels have been drawn between the neuropsychological performance of alcoholics and other patient groups with well documented brain damage in specific locations.

One problem with the approach taken to this point, in organizing the alcoholism findings around the impairment of specific brain structures, has been that

the conclusions reached have been based solely on inference. In other words, the procedure followed has involved establishing the psychological correlates of lesions to particular brain sites from the more general neuropsychological literature, then showing similar psychological patterns in alcoholics, and finally, inferring that the pattern of neuropsychological deficits found in alcoholics was a consequence of the same neuropathology. Admittedly, this line of reasoning has received some support, particularly in the case of the anterior-basal hypothesis, from the finding of the corresponding type of neurological deterioration in alcoholics that mediates the behavioural impairments in question in other brain damaged groups.

What the investigations reviewed fail to do, is to demonstrate the correlation between neurological impairment and psychological deficits within the same alcoholic population. The dangers of inferring brain damage, or even more pertinent, a particular type of brain damage, from similarities between psychological performance in one diagnostic group and another group with known brain damage, are legion. One difficulty is that factors other than neuropathology can produce deficient performance on neuropsychological tests. This point was made earlier in this chapter with regard to the effects of aging and alcoholism. It is also an issue that has been of major concern in the study of cognitive functioning in schizophrenics.

Fortunately, in the alcoholism field, there are a few studies where neurological and psychological data have been collected from the same patient sample. Relevant here are two studies by Willanger (Willanger et al, 1968; Willanger, 1970), demonstrating a high correlation between the presence of cortical atrophy and ventricular dilation with clinical and psychometric evidence of cognitive dysfunction.

Parsons (1977) cites three further relevant investigations. The first (Haug, 1968), already mentioned above, divided alcoholics into two groups; one with evidence of personality and/or intellectual deterioration, the other without these signs of psychological deficit. All patients were subsequently tested by pneumoencephalography Nineteen out of the 20 patients in the deteriorated group showed evidence of ventricular dilation. In the nondeteriorated group, 15 out of 31 evidenced similar dilation. This difference was significant at the .01 level.

The second study (Ferrer , 1970) assessed neuropsychological impairment with the Grassi Block Substitution Test and the Minnesota Perceptuo-Diagnostic Test. They also took pneumoencephalograph recordings from the same group of patients and rated degree of brain atrophy on a four point scale. The correlation between the neurological and psychological measurements was .68, with greater psychological dysfunction associated with greater brain atrophy.

The final study (Brewer & Perrett, 1971) was similar methodologically to Ferrer et als' investigation, although different neuropsychological measures were used (the Benton Visual Retention Test and the WAIS). Measures of cortical atrophy, ventricular atrophy, and the width of the third ventrical, all showed moderate positive correlations (ranging from ·31 to ·52) with results from both of the psychological tests.

This work shows that there is indeed a relationship, at a gross level, between brain damage and neuropsychological performance in chronic alcoholics. However, this literature is yet to establish neuropsychological correlates for brain damage confined to specific brain loci. In other words, it does not yet address the issue of the particular forms of brain pathology that underlie the major forms of neuropsychological deficit that appear to characterize many alcoholics.

From the size of the correlations in the studies cited, it is evident that much of the variance is not explained by the relation of the two sets of measures. This provides a cautionary note to the assumption that cognitive impairment associated with alcoholism is caused only by organic pathology. However, the relationship might be found to be greater if tests were selected on the basis of some theoretical rationale, for example because they are known to be sensitive to a particular form of brain pathology, and then performance on these tests related to the organic state of that particular

brain site. Hopefully, the next generation of research will be less atheoretical and based on a more refined measurement technology. Fortunately, continued empirical and conceptual advances in neuropsychology, the widespread availability of multivariate statistical packages, and the development of highly sensitive neurological procedures including the EMI Scanner, EEG spectral analysis, and computer-assisted methods of evaluating EEG evoked potentials, augur well for the future.

Reversibility of Neurological and Cognitive Impairment.

The question of whether or not the cognitive deficits demonstrated in alcoholics during the first few weeks or months of abstinence are permanent is an important one. Apart from being of obvious concern to recovering alcoholics, the answer to this question has important implications for models proposed to account for cognitive dysfunction in alcoholics, as well as for considerations of the possible relevance of these deficits to treatment participation and post-treatment functioning. Given the importance of this issue, it is surprising how few well designed experiments have directly investigated the recoverability of psychological functions following the cessation of drinking.

Although few methodologically adequate studies have addressed the question of psychological recoverability, much less has been done by way of more direct neurological investigations to assess changes that might occur in the

organic condition of the brain following sustained abstinence. Indeed, Ron (1977), in his extensive review of the neurological literature, could only locate one relevant study. This was by Ledesma Jimeno (1958) who repeated pneumoencephalograph testing in five alcoholics after periods of abstinence ranging from two months to two years. Over this period, degree of cerebral atrophy remained unchanged. Other major reviewers appear to have had similar difficulties. The only other relevant study to emerge being an EEG investigation by Bennett et al (1960) in which initial abnormal tracings were found to change toward normality after a period of abstinence. Unfortunately, this finding is not helped by the knowledge that conventional EEG analysis is not particularly sensitive to alcohol-related brain damage, even in the advanced stages of Korsakoff's Psychosis and Alcoholic Dementia (Cutting, 1978b). Consequently, nothing of any substance can be concluded regarding the biological integrity of the brain as a function of continued abstinence. No pun intended.

At a number of points throughout this chapter, indications of improvement in some areas of cognitive functioning have been noted. For example, a fairly extensive account was given of decreases in field dependence. Also, on some tasks (e.g. paired-associate learning), studies showing contradictory findings were cited. Because experiments conducted within the first two weeks of hospitalization generally yield impairment on this type of task, whereas those conducted later fail

to find impairment, it seems reasonable to postulate that the discrepancies might have arisen because of improvements in secondary verbal memory over time in treatment. It is worth noting however, that on a variety of other tasks administered to alcoholics after two or three months of abstinence, impairment was still evident.

Although adequately controlled studies are sparse, there are a large number of reports of repeated test administration to patient groups. In the main, these repeated assessments have been confined to the first 10 weeks of abstinence. Tasks on which improvements have been shown over this period include: WAIS Subtests (Page & Linden, 1974; Clarke & Haughton, 1975), Perceptual-motor tasks (Tarter & Jones, 1971), Rod and Frame Test (Chess et al, 1971; Goldstein & Chotlos, 1966; McWilliams et al, 1975; Smith & Layden, 1972), Embedded Figures Test (O'Leary, 1977; Smith et al, 1971); varied learning and memory tasks (Allen et al, 1971; Berglund et al, 1977; Page & Linden, 1974; Weingartner et al, 1971), motor tasks (Goldstein , 1976 ; Tarter & Jones, 1971), abstraction tasks (Ornstein, 1977; Page & Linden, 1974; Smith et al; 1971), and Hooper's Visual Organization Test (Ornstein, 1977).

These studies suggest that some improvement does take place in a number of important psychological and behavioural capacities during the first few months of abstinence. However, demonstrations of complete recovery are rare. Generally, impairments are still found on the

the final test occasion. In most studies, these impairments are of sufficient magnitude to suggest mild to moderate degrees of organicity. The results of these and other investigations further suggest that most recovery takes place within the first few weeks and from then on, further improvement is either slight or nonexistent. For example, in one of the studies already cited (Clarke & Haughton, 1975), alcoholics and matched controls were tested at two, six, and 10 weeks after withdrawal. Some improvement occured in the alcoholic group during the first six weeks, but thereafter, further change was minimal. It is also of interest that at 10 weeks, the alcoholic group was still significantly more impaired than the control group on measures of psychomotor speed and abstract reasoning. Other studies, using a variety of neuropsychological tests, suggest that the period of maximal recovery might in fact lie within the first two weeks, and from that time, recovery follows an asymptotic curve (Page & Linden, 1974; Allen et al, 1971; Jonsson et al, 1962; Carlsson et al, 1973).

Unfortunately, with the exception of Page and Lindens' (1974) experiment, all of the studies mentioned up to this point have failed to control for the practice effects that frequently occur when tests are administered repeatedly. This is an important consideration. After all, it is conceivable that the apparent improvements have been a consequence of this factor, rather than 'true' changes in cognitive capacity. Certainly, it has been

demonstrated that practice can help to overcome the adverse effects of alcohol on the performance of a number of perceptual, motor, and cognitive tasks (e.g. Goldstein , 1976; Tarter, 1971, 1976).

The two studies that have included adequate controls for practice effects (Page & Linden, 1974; Goldman & Rosenbaum, 1977), in the main, show similar results to other investigations in this area. Maximal improvement generally occurs after the first two weeks and thereafter, change is limited. Significant practice effects were not evident on the measures used, thereby lending support to the inferences drawn from the less adequately controlled studies.

Page and Linden (1974) administered a comprehensive test battery, including the WAIS, the Trail Making Test, and Benton's Visual Retention Test. Their findings are of particular interest because in spite of improvements, on the final testing after eight weeks of abstinence, deficits of a similar type and magnitude to those described in the seminal Fitzhugh et al (1960, 1965) papers were still present.

Goldman and Rosenbaum (1977) measured a variety of learning and memory dimensions. Like numerous other investigators, they found no deficit in vocabulary knowledge at any time during treatment. On two indices of ability to learn new verbal material, they found severe impairments on the fifth day of inpatient status. In contrast on the fifteenth day they found a return to normal levels, in

both the group previously tested as well as in a second group tested for the first time. A similar level of performance was evident in three groups tested at 25 days. In contrast to these findings, on a measure of visuo-perceptual learning (Stark's visuoperceptive subtest), they found severely impaired performance at five days, and, at 15 and 25 days, some recovery among subjects with short drinking histories. However, for those subjects with alcoholic drinking histories of over 12 years, performance remained at a level of impairment that was not significantly different from day five.

The results of the study just described are of interest because they suggest that there might be different recovery rates for particular types of cognitive dysfunction. Further, they imply that deficits in some areas might be more permanent and that this lack of reversibility may be in part a function of length of alcoholic history. These possibilities provide potentially fruitful leads for future research.

The studies discussed in this section have only looked at changes that occur during the first few weeks or months of abstinence. Although a number of these studies suggest a leveling off in improvement rates by the final testing, sometimes within the normal range although more typically, still at dysfunctional levels, they do not tell us anything about longer term changes after alcoholics leave treatment programmes.

Two studies have addressed the question of cognitive functioning in alcoholics who have had long periods of abstinence prior to testing. Templer et al (1977) administered the Trail Making Test to alcoholics who had been abstinent for periods ranging between one to 14 years and Greiner (1961) tested aspects of short-term memory in alcoholics who had had from two to 11 years of sobriety. Both studies failed to find evidence of cognitive dysfunction. Their respective authors considered that the results they reported suggested evidence for long-term recoverability of intellectual functioning. Without additional testing at an earlier time however, this conclusion is unjustified. The major problem here is that it cannot be assumed that the alcoholics tested in these studies constituted representative samples of alcoholics who show signs of neuropsychological impairment while they are in hospital. In particular, if the major hypothesis under investigation in this thesis has any validity, namely that cognitive dysfunction is a major factor in the prediction of post-treatment relapse, then it could be expected that those individuals who sustain an abstinent outcome for such long periods of time will be less impaired.

Fortunately,there are a few studies in the literature that have employed repeated assessment. Supporting the position of long term reversibility is a study by McLachlan and Levinson (1976). These researchers found improvement on the WAIS Block Design Subtest over a one year period of abstinence, to the point where no impairment was

detectable. Again with a one year retesting interval, but using a wider variety of tests, Long and McLachlan (1974) showed significant improvement in a group of 17 abstinent alcoholics. Improvement was found on several WAIS subtests, the overall Impairment Index of the Halstead-Reitan Battery, the Finger Tapping Test, and the Category Test. No improvement was found on the Trail Making Test however, and on the Digit Symbol and Block Design Subtests of the WAIS, their improved scores were still significantly below the control group.

O'Leary et al (1977) also conducted long term testing. They administered the Trail Making Test to a group of alcoholic patients who had been abstinent for between nine to 14 days and to a group of carefully matched nonalcoholic controls. Followup testing was conducted 12 to 16 months later. They found that the control group performed at the same level on the second administration as they had on the first. In contrast, the alcoholics (both a group that had abstained and a group that had relapsed) improved significantly over time on Part B of the test. This final performance still placed them significantly below that of the control group.

The only other study that appears to have looked at long term recovery is by Berglund et al (1977). Alcoholics who had been tested during a previous hospitalization were retested approximately two to six years later. Both abstinent and relapsed patients were retested, although none had been drinking during the two weeks prior

to the second testing. At followup, the subjects were divided into improved and unimproved groups. The improved subjects had consumed either none or slight amounts of alcohol since the first testing. The unimproved subjects had had a large number of relapse periods. At the initial testing, the alcoholic subjects were significantly more impaired than a matched group of nonalcoholic subjects. Both improved and unimproved alcoholic subjects were found to have improved their performance at the second testing on a verbal paired-associate learning task and on Benton's Visual Retention Test. On one of the measures, Koh's Block Designs, only the improved group performed at a significantly superior level on the second test occasion. Neither group improved on a fourth test, the Colour Word Test.

Berglund et al did not provide data on the nonalcoholic controls. Consequently, an assessment of the levels of performance at the second testing, relative to nonalcoholic subjects, could not be made. The finding of improvement on some measures, in both the group that had continued drinking and in the improved group, is common to the last two studies reviewed. This suggests that these changes could not be explained only by reduced alcohol intake. It also raises the question of whether improvements found in other studies have been due solely to abstinence. It might be that reduced anxiety due to habituation to the testing situation, or other factors such as improved motivation or practice effects are also important. Once again, we are cautioned in making the assumption that

performance on neuropsychological measures are only, or even primarily, sensitive to the organic state of the brain.

In conclusion, from the data available, it appears that many, but not all psychological and behavioural capacities, show improvement following a period of abstinence. On a number of tasks, most improvement occurs within the first two weeks. From then on recovery is slower, although the rate apparently varies depending upon the psychological function involved. In some cases, long term recovery is complete, in others it is either partial or absent. There is some evidence that suggests more advanced alcoholics, possibly those with more extensive neurological damage, show a diminished capacity for recovery. However, this question, and the relation of cognitive recovery to neurological damage more generally, as well as to other factors, has yet to be directly addressed.

Causes of Brain Damage and Cognitive Dysfunction.

Both the presence of brain damage and the presence of a variety of forms of cognitive dysfunction have been found in association with alcoholism. Given this association, it is often assumed that(a) alcohol itself causes brain damage, and that (b) alcohol causes cognitive deterioration, probably via the damage caused to brain structures. The logical fallacy involved in making inferences of causation from demonstrations of association

is of course frequently heralded.

At present, the etiology of brain damage and neuropsychological impairment among alcoholics is not known. However, many suggestions have been advanced. Examples include: the direct toxic effect of ethanol (Freund, 1973), the indirect effect of alcohol via disruption to liver functioning (Smith & Smith, 1977), deficient nutrition (Victor & Adams, 1961), and head injuries (Goodwin & Hill, 1975). Another possibility, already discussed in relation to RFT performance, is brain dysfunction prior to the onset of alcoholic drinking (see also Tarter, 1976).

One issue that bears on the question of etiology, is the more detailed relationship that has been suggested between consumption variables and cognitive functioning. For example, although the vast majority of neuropsychological studies of alcoholics have found evidence of dysfunction, one study (Tarter et al, 1975), failed to find any evidence of impairment on an extensive battery of 23 tests. The authors concluded that this discrepant outcome arose because of the short duration of alcoholism within their sample (mean = 7·2 years) relative to populations used in other studies. In the studies reviewed previously, some showed significant correlations between years of alcoholic drinking and severity of neuropsychological impairment. Very few studies, unfortunately, have corrected for age effects. One that did (Jones & Parsons, 1971), found that with the effects of age partialled out, long-term alcoholics

were still significantly more impaired than short-term alcoholics. In the last section, some support was indicated for the view of diminished potential for cognitive recovery following abstinence in long-term alcoholics.

In a few studies already cited, it was shown that although performance on some tasks correlated inversely with years of alcoholic drinking, performance on other tasks (e.g. some short-term verbal memory tasks and RFT) failed to show this relationship. This suggests that separate cognitive functions (and presumably separate brain mechanisms) might be differentially influenced by different parameters of drinking history. This possibility has only been investigated very recently, both in social drinkers (Parker & Noble, 1977) and in alcoholics (Eckardt et al, 1978).

In a sample of social drinkers, Parker and Noble (1977), failed to find significant relationships between total lifetime alcohol consumption and cognitive dysfunction or between current frequency of drinking occasions and cognitive dysfunction. However, in contrast to these findings, impaired performance on the Wisconsin Card Sorting Task, the Shipley-Hartford Scale, and the Halstead Category Task, showed highly significant age-partialled correlations with the current quantity of alcohol consumed per drinking occasion. Indices of verbal memory, learning, and organizational processes were not significantly correlated with any of the drinking variables. When the sample was dichotomized into light and heavy social drinkers

however, within the heavy intake group, a significant correlation explaining 26 percent of the variance emerged between current quantity consumed and verbal learning impairment. The authors concluded that even moderate social drinking is related to impairment in high level cognitive processes including adaptive ability, concept formation, and the capacity to shift from one idea to another. They also concluded that heavy social drinking extends the range of functions involved to memory and learning deficits.

Eckardt et al (1978) conducted a similar study to that just described. In contrast however, they used a sample of 95 male alcoholics between the ages of 21 to 60. Subjects were administered 17 neuropsychological tests within seven days of their last drink. Overall, the group's performance on measures of abstraction, memory, and concept formation, was within the range of impairment typical of many studies previously reviewed (e.g. Fitzhugh et al, 1960, 1965). It was found that cognitive performance did not vary as a function of any of the following independent variables: prior or current physical or health conditions, head trauma, history of seizures, DT's, blackouts, sleep disorders, shakes, or missed meals in the last six months because of drinking.

In Eckardt et als' study, relationships between drinking variables and cognitive performance were assessed by correlational analyses, with the effects of age and educational level partialled out. Neither date

of last drink or frequency of drinking occasions during the last six months was correlated with any of the cognitive scores. Those measures that did show significant correlation were retained for subsequent multivariate analyses. Next, linear multiple regression analyses were run using age, education, and various combinations of recent and chronic consumption indices as independent variables. The 17 cognitive measures were the dependent variables. Further analyses included, in addition to the first-order terms, interaction terms (cross products formed from pairs of independent variables), and quadratic terms. The best equations from these analyses were more powerful predictors of cognitive dysfunction than were the linear equations. The amount of variance explained (R^2) varied considerably (between two and 37 percent) from one cognitive task to the next.

The above analyses showed that performance on some cognitive tests could be predicted from consumption variables, whereas others could not. Within the group of tasks that were related to previous drinking practices, some were found to be related only to chronic drinking (e.g. the Tactual Performance Test - Memory, the Trail Making Test, Benton's Visual Retention Test, and the Shipley-Hartford), whereas others were found to be associated with recent consumption (the WAIS Object Assembly Subtest and the Seashore Rhythm Test). The authors speculated that performance on tests related only to recent drinking patterns might be more reversible whereas performance on tests linked to chronic consumption

might be more resistant to recovery following sustained abstinence.

In the study under discussion, as in Parker and Nobles' sample of social drinkers, frequency of alcohol consumption was not inversely related to cognitive performance, whereas average amount of alcohol consumed per drinking occasion was so related. This raises the not too surprising possibility that disruption to brain functions only occurs once a certain blood alcohol level is attained. In contrast to the former study however, chronic alcohol consumption (both years of alcoholic drinking and total life-time consumption) did predict degree of cognitive dysfunction on a number of tests. Because alcohol consumption does appear to have a cumulative effect, it may be that changes of this type in social drinkers, if present, are too subtle to detect. Alternatively, they might be present, but so small in magnitude that they are swamped by acute effects.

One of the more interesting findings of Eckardt et als' investigation was the presence of significant interactions between chronic and recent consumption measures in predicting cognitive performance. These interactions indicated that beyond a given point on either of the two chronic measures, if consumption per drinking occasion over the last six months also went beyond a certain magnitude, then strong predictions could be made regarding degree of cognitive dysfunction on a majority of the tests. Below this 'critical value', only very weak

or nonsignificant predictions could be made. This, considered in relation to the finding of curvilinear terms in a number of the optimally predictive equations, suggests that certain patterns of drinking behaviour will accelerate changes in cognitive functioning that initially occurred at a slower rate. Although not commented upon by the authors, the common occurrence within the equations of significant interaction terms formed between age and intake measures, further suggests that the aging brain might be particularly sensitive to the effects of alcohol abuse.

The authors of both of the studies just discussed, considered that their respective findings support claims that excessive alcohol intake plays an important part in the development of cognitive dysfunction. Other findings that are consistent with the view that alcohol intake per se is implicated in alcoholism-related cognitive impairment, include the following: (1) a variety of parallels have been demonstrated between acute and chronic effects of alcohol abuse; (2) a number of studies have shown an inverse relationship between chronic consumption and performance on a range of cognitive tasks after varying periods of abstinence; (3) animal experiments, where alcohol intake was the only independent variable manipulated, have produced both acute and permanent deficits in learning and memory capacity (e.g. Freund, 1973, 1975);

(4) other animal experiments have shown that alcohol and/or its metabolites, can produce neuropathology (Freund, 1973). Other studies, including Eckardt et al (1978),

have shown that factors such as history of head injuries, and dietary neglect, are not associated with many forms of cognitive dysfunction found among alcoholics.

Although the findings assembled in the last paragraph provide some corroboration for the view that alcohol itself is an important factor in the production of brain damage and cognitive dysfunction in alcoholics, they do not prove that alcohol causes this deterioration in humans. This is certainly a possibility, but even if it is so, the effect may be mediated indirectly by metabolic disturbances in other parts of the body, for example the liver (Smith & Smith, 1977). Further, it has already been established in this chapter that the association between brain damage and cognitive dysfunction in alcoholics appears to be of only moderate strength. However, it was also noted that this relationship could be expected to increase if more refined measures were used and if their selection rested on a firm theoretical rationale. Additionally, even when a comprehensive list of intake measures are included in regression models that maximize their effects, they only account for a minority of the variance on neuropsychological tests. Further, performance on some measures are not predicted by alcohol intake, either acutely or chronically. The best example here is Field Dependence as operationalized by RFT. Finally, although probably not important in many areas of cognitive functioning, dietary neglect has been strongly implicated in certain forms of alcohol-related neuropathy, particularly that associated with Korsakoff's Syndrome.

Most of the studies bearing on the issue of the etiology of alcoholism-related cognitive impairment have

been correlational in design. Although this approach has value in disconfirming hypothesized relationships, for example, that head injuries typically associated with alcoholism are major factors in the genesis of cognitive dysfunction, it does not provide a basis for strong causal inference. In large part, this is because it does not indicate the direction of a causal relationship. This is an important consideration with regard to the question under discussion. For one thing, it might be that rather than showing dysfunction because they drink too much, alcoholics may drink too much because they manifest dysfunction. In other words, their brain damage and/or cognitive impairment may antedate their alcoholism.

In recent years, some support has in fact arisen for the view that at least some, and possibly many alcoholics have minimal brain damage and/or the hyperactivity syndrome, as children. It is not clear how closely these two conditions coincide although, many of the characteristics defining the hyperactivity syndrome are also seen as signs of slight to moderate central nervous system pathology.

It was mentioned in chapter one (page 15), that the children of alcoholic parents have a much greater probability of becoming alcoholics as adults than children of nonalcoholic parents. This finding also holds, although not so strongly, when the progeny of alcoholics are raised apart from their biological parents. It is also known that alcoholic parents have a much greater than chance

probability of having children who are diagnosed as hyperactive and/or as being minimally brain damaged (MBD) (e.g. Morrison & Stewart, 1973a, 1973b). A genetic interpretation is favoured here, as well as with the children of alcoholic parents who become alcoholics, by the demonstration that the frequency of these diagnoses in children raised by their biological parents is similar to the frequency found when they are raised by adoptive parents (Goodwin et al, 1975; Morrison & Stewart, 1973a).

An important question arises from the findings outlined in the previous paragraph, namely, are the children who become alcoholics the same children who are diagnosed as hyperactive or MBD? Some support for synonymity or large overlap between the two groups, comes from a followup study of MBD children. In a sample of these children, by the ages of 12 to 16, fifteen percent were already showing signs of excessive drinking (Mendelson et al, 1971). Further corroboration comes from retrospective studies of alcoholics. For example, Tarter (1976) found that severe drinkers reported four times as many MBD symptoms as children than less severe drinkers. It is also of interest that they evidenced less psychopathology, as measured by the MMPI, than the less severe drinkers. It is of further interest that the MBD syndrome in childhood has been shown to be related to the development of hysterical disorders and psychopathy in the adult years (e.g. Guze, 1975).

From the studies just reviewed, there is the

possibility that some genetically transmitted factor manifests itself phenotypically as MBD in childhood and subsequently, in adulthood, increases the likelihood that alcoholism and/or sociopathic behaviour and/or an hysterical disorder will occur. This line of research is still in its infancy. Research workers have yet to identify the nature of the brain damage and cognitive dysfunction that accompanies this trait or constellation of overlapping traits. However, the findings to date do raise the possibility that some of the brain damage and/or cognitive impairment found among alcoholics in treatment settings may antedate their excessive drinking patterns.

Arguing against the view that all damage found among alcoholics antedates their alcoholism is the observation that not all alcoholics, indeed not even the majority, appear to have MBD symptoms during childhood - although this point needs to be more carefully investigated. Related to this is the finding that many patients who show obvious signs of deterioration, failed to show evidence of similar impairment prior to their alcoholism or during the early years of their alcoholic history. Additionally, there is the common finding that 'hold' tests (those presumably measuring crystallized intelligence) are significantly better performed than are measures sensitive to brain dysfunction (those presumably indexing fluid intelligence). This suggests an aquired deficit. Or, putting it another way, a drop from previous cognitive levels. A related point that appears to have been overlooked, is the common finding that sociopathic

individuals typically have a pattern of intellectual performance that is the reverse of that just described (see Trasler, 1973 for a review). That is to say, their fluid intelligence level is frequently significantly higher than that of their crystallized abilities. This is generally considered to occur because their childhood behaviour pattern prevented them from realizing their intellectual potential in school pursuits and subsequently, in occupational attainment. This interpretation is favoured over socioeconomic deprivation because of the finding that the crystallized-fluid discrepancy is even geater among individuals from'superior' homes than those from working class families (Manne et al, 1962).

In conclusion, it is clear that the question of the origin of cognitive impairment and brain pathology among alcoholics is a complex issue. With regard to some areas of cognitive functioning, there is evidence supporting the view that impairment antedates alcohol abuse, for example Field Dependence as measured by RFT. In most areas, the bulk of the empirical data seems to favour the view of acquired impairment, probably induced by certain patterns of prolonged alcohol abuse although, at least in some instances, resulting from alcoholism-related dietary neglect. This interpretation is complicated by the apparent over-representation among alcoholics of individuals who were minimally brain damaged as children. For many alcoholics, it may be found from future research that both interpretations are correct. That is, some deficits may precede the onset of their alcoholic drinking

pattern. However, once this pattern is underway, further impairment may be induced in the areas already deficient. In addition, the range of functions impaired may increase over time. To clarify the patterns of etiology will necessitate detailed longitudinal tracking and assessment from childhood into the middle or late adult years. Those who have engaged in followup work with alcoholics will appreciate that this is a tall order. However, until it is accomplished, we are left with indirect evidence and inference as our only tools to untangle this complicated and important question.

Conclusions.

Evidence for the presence of both neurologically defined brain damage and a variety of neuropsychological deficits among alcoholics in treatment settings is compelling. It appears that somewhere between five and 10 percent of alcoholics seeking treatment have deterioration of sufficient magnitude to justify the diagnosis of a chronic organic brain syndrome, either Alcoholic Dementia or Korsakoff's Psychosis. Probably somewhere over 50 percent of the remaining patients possess varying degrees of pathology that is detectable by extant methods of neurological and neuropsychological measurement. Because of variable degrees of improvement found on some neuropsychological tests, this figure will probably be lower if assessments are made after longer periods of abstinence.

In making these statements, it is recognized that they have not been proven. This is because for the strict logician, only false conclusions (predictive failures) can be considered to bear conclusively on a premise. Successful predictions, in contrast, carry a small information content. What is concluded here is that the statements in question have frequently survived tests of falsification. Or, in Popper's terminology, they have been extensively corroborated (Popper, 1972). Because they have survived this test often, confidence in the hypothesized relationships has increased to a high level. To put it crudely, although accurately, we 'feel' that they have validity. In the final analysis, this is the best we can do in any field of scientific endeavour, and it is upon this 'feeling' that we take further action, whether we be researcher or clinician.

Having made this cautionary statement, let us continue with our overview of the 'findings' or 'tentative conclusions' that exist in this field.

Although the presence of organic and cognitive deterioration is a consistent finding, at least within populations where a majority of patients have alcoholic histories extending beyond 10 years, knowledge of the mechanisms underlying the observed behavioural pathology is less well advanced. Regarding organic impairment, the neurological data suggests that the most common form of brain damage is a mild to moderate degree of diffuse cortical atrophy and microscopic structural changes,

associated with more pronounced deterioration in prefrontal areas, in some cases extending into subcortical limbic structures. Also apparently relatively common, and frequently accompanying frontal damage, is a focus of more extensive atrophy in the parietal region.

A number of the deficits found in psychological functioning are consistent with the pattern of deficits that have been shown to accompany anterior-basal damage in nonalcoholics. Inconsistent with the neurological finding of diffuse cortical damage, is another common pattern of neuropsychological deficits - a pattern that is strongly suggestive of right hemisphere involvement, particularly in the right parietal region. Behaviourally, this pattern is mainly expressed in deficient performance on a wide variety of tasks that involve spatial perception or integration. It was suggested that rather than reflecting more advanced organic deterioration in the right hemisphere, these findings probably indicate that these functions are more readily disrupted than the predominantly verbal left hemisphere functions.

Overall, although there are quite strong parallels between the neurological and psychological deficits, suggesting that the former causally mediate the latter, the correspondence is far from being one-to-one. In other words, brain damage is not the only determinant of cognitive dysfunction amongst alcoholics.

There are also some parallels between the psychological performance of alcoholics and elderly persons, suggesting that at least in some ways, the behavioural sequelae of alcoholism can be viewed as a form of premature aging. This ties in with the finding that alcoholics perform well on overlearned, verbally-mediated behaviours built up over the years, in contrast to often dramatic deficits on tasks requiring behavioural flexibility and new learning that does not draw heavily on past experience.

Poorly understood as yet is the degree of reversibility that exists in neurological and psychological mechanisms. After a prolonged period of abstinence, some cognitive deficits, at least among alcoholics with shorter drinking histories, appear to be completely reversible. Others seem to be either partly reversible or irreversible.

The causes of cognitive dysfunction and alcoholism-related neuropathy, other than in the well defined neurological clinical syndromes, are also poorly understood, although it does seem that particular patterns of alcohol abuse sustained over a number of years are important antecedents of impairment. This issue is complicated by the possibility that a least certain types of deficit seem to predate the onset of alcoholism.

CHAPTER THREE

THE SIGNIFICANCE AND CLINICAL IMPLICATIONS OF BRAIN DAMAGE AND COGNITIVE DYSFUNCTION

Introduction.

Arising out of the neurological and neuropsychological literature reviewed in the last chapter, are a number of points that seem particularly relevant to the task of increasing our understanding of alcoholism and its treatment.

In chapter one, it was concluded that attempts to find a personality pattern unique to alcoholics has not been successful. Goldstein (1976) has suggested that the neuropsychological findings raise the possibility that a pattern of cognitive dysfunction might be identified that is relatively unique to alcoholics and that this would be useful diagnostically. This may be so and indeed, to a limited extent, it would seem that this has already occurred. However, this enterprise clearly falls within the tradition of unitary approaches to alcoholism. It was argued earlier that this way of conceptualizing alcoholism research and treatment carries with it major shortcomings. Further, and in contradiction to this way of thinking, a major feature of the cognitive and neurological literature has been the demonstration of heterogeneity within alcoholic populations, both in the type and extent of dysfunction found.

A more fruitful application of the neuropsychological

approach and findings to date, may lie in extending our knowledge of the psychology of alcoholism. In other words, using this body of knowledge to provide empirically based explanations to many of the issues addressed earlier by the unitary model. For example, in understanding mechanisms that might underlie "loss of control" or "inability to abstain" and other aspects of response to treatment and relapse processes.

The Role of Neuropsychological Dysfunction in the Development of Alcoholism.

The findings relating childhood MBD to the development of alcoholism in adulthood provides a potentially valuable lead to the question of why at least a subgroup of alcoholics follow that particular "career path". The suggestion that high Field Dependence might also predate the onset of drinking problems in many alcoholics also has potential relevance to the question of etiology. Although the question of why certain individuals become alcoholics when others who drink heavily do not, is vitally important and carries with it implications for future prevention, it will not be taken further at this point. The reason for this being that the main concern of this thesis lies with what happens after people have developed drinking problems and, in many instances, developed cognitive impairment. From this vantage point, the original cause of cognitive dysfunction is of little consequence. Of major concern, however, are the effects that the presence of this impairment

has on subsequent behaviour, behaviour which includes attempts to make alterations in previous drinking patterns and life styles.

Neurological and Cognitive Impairment in the Understanding of Alcoholism.

This section is concerned with the possible relevance of the literature reviewed in chapter two to a number of important features of alcoholics observed in treatment settings. One area addressed is the relation of this work to what can very losely be defined as the "alcoholic personality". Other major areas include the possible implications of the neuropsychological findings for treatment participation and outcome.

The Neuropsychological Capacities of Alcoholics in Treatment Programmes.

Briefly, let us review the pattern of deficits that has been demonstrated in a substantial number of alcoholics participating in treatment programmes. A consistent finding has been the lack of impairment in older, well established, primarily verbal skills. On the other hand, throughout the period that alcoholics are typically in treatment (the first one to three months after detoxification), a large percentage have been shown to be deficient on a variety of tasks requiring new skills or problem solving, particularly those that require visuospatial and

abstracting behaviour. These deficits are considered to indicate a decrease in fluid or biological intelligence. Decline in these aspects of intellectual functioning has also been demonstrated in the very elderly. Intact fluid intelligence is considered to be particularly important in making adaptive adjustments to changes in the environment and in aquiring new skills. Biological intelligence is considered to include a group of high level skills which include planning, foresight, and decision making. Deficits in both fluid and biological intelligence are seen generally as leading to reduced overall flexibility. There appears to be a parallel here with field dependence in that individuals with high field dependence have difficulty in coping with unstructured or ambiguous perceptual and social situations, although alcoholics with high field dependence do not always show severe deficits in fluid or biological intelligence.

Apart from diffuse cortical deterioration, a high proportion of brain damaged alcoholics appear to have a major focus of atrophy in the frontal cortex and related subcortical structures. Much of the observed impairment on tasks presumed to reflect biological intelligence appears to arise from damage to this area. Nonalcoholics with this type of brain damage have been observed to be easily distracted, to have difficulty in changing from one activity to another or in doing things, conceptually and behaviourally, in different ways. They also tend to be deficient in using errors as corrective information. These observations seem to parallel the types of neuropsychological

test deficits typical of many alcoholics.

Frontally impaired individuals are also frequently severely deficient in their ability to picture themselves in relation to future events as a potential agent. Possibly related to this is the finding that they have difficulty in verbally controlling ongoing behaviour. Putting it somewhat loosely, there is a lack of integration between their intentions and their actual performance. They are also claimed to often make decisions without sufficient information. Whether this is due primarily to aspects of their diminished cognitive capacity or to temperamental changes, for example impulsivity, is not clear. Other personality changes have also been observed. These include increased extraversion, reduced social inhibitions, and reduced anxiety. However, although common in frontally damaged individuals, these personality changes have not been sufficiently investigated in alcoholics with frontal deterioration to justify statements regarding their prevalence.

Neuropsychological Impairment and the 'Alcoholic Personality'.

Ron (1977) considers that a number of the behavioural symptoms of frontal lobe impairment could be interpreted, or more correctly misinterpreted, as part of the make up of the so-called alcoholic personality. By this, he means that characteristics such as 'inability to abstain' may relate more to changes following from frontal damage, for example, decreased self critical capacity, reduced ability

to regulate ongoing behaviour, or learning from experiential feedback, rather than stemming from some intrinsic property of the 'alcoholic personality' such as the 'death wish' of the analytically oriented theorists.

To explore in detail the possible relationship between cognitive dysfunction and personality necessitates venturing into the literature pertaining to personality functioning among alcoholics. This is a vast literature. It is also a very confused literature. Fortunately, in recent years, some order has started to emerge.

Attempts to validate ideas about the so-called 'alcoholic personality' (e.g. oral fixation and subsequent behavioural dependency) have proven to be fruitless exercises. Many of these studies, particularly the earlier ones, utilized projective devices. Unfortunately, on those occasions where the findings were interpreted as corroborating a particular theoretical model, the protocols were frequently not scored blind. As a consequence it is often unclear whose projections were being assessed, the alcoholics' or the researchers'. In the main, this work has shown that there is no one 'alcoholic personality'. Nevertheless, it is interesting to consider the generalizations that have been made on the basis of the projective literature. For example, Neuringer and Clopton (1976) list the following generalizations from Rorschach data: "(1) Alcoholics are more psychopathic in nature than neurotics. (2) They show an incapacity to tolerate stress. (3) They lack the perseverance to overcome difficulties.

(4) They are grandiose in their plans but do not have the patience or the concentration ability to reach their goals. (5) Alcoholics have a higher level of guilt and anxiety than does the psychopath but they show fewer of these feelings than do neurotics. (6) They are egocentric and lack emotional depth and warmth. (7) Alcoholics have poor or superficial interpersonal relationships. (8) They tend to be constricted, think in stereotypes and are pedantic. (9) Alcoholics use regression as their major defence mechanism" (p. 12).

Although the presence of some of the characteristics listed above have not been validated subsequently, and others have been shown to apply only to a subgroup of alcoholics, they do bear considerable resemblance to a number of the features noted in the neuropsychological literature. In particular, there appears to be a parallel between some of these characteristics and changes that are presumed to stem from frontal lobe dysfunction, thereby providing supportive data for Ron's (1977) suggestion that so-called personality traits may in fact be sequelae of organic impairment.

In contrast to the earlier studies, more recent work has been based on the assumption that there is no constellation of traits associated exclusively with alcoholism. Consequently, these investigations have tended to drop preconceived theoretical notions and assume a more atheoretical, empirical orientation. Most studies

have relied on so-called objective test procedures, for example the 16PF and the MMPI.

The findings of the research based on objective test batteries do not deny the presence of many of the characteristics observed in the projective literature. What this literature does show, is that like the neuropsychological findings, some characteristics are found in many alcoholics. However, they cannot be generalized to all alcoholics in treatment settings. Examples of common traits in hospitalized alcoholics include psychopathy and depression. Consistent with clinical observations of these two types of pathology is the frequently replicated finding that the most common MMPI profile includes elevations to scales two and four. A number of studies have identified fairly clearly definable subtypes of personality profiles among alcoholics, indicating that the pooling of data across all alcoholics in a given sample can obscure important within population heterogeneity.

Although the number of personality types that are identified depends largely on how fine-grained the researcher makes his or her analysis, a recent reviewer of this literature (Kurtines et al, 1978) concluded that recent multivariate investigations have shown a remarkable convergence of results, a convergence all the more compelling given the wide range of assessment procedures and methodological approaches used. Two frequently occurring

subtypes are described as: (1) extremely low impulse control, impulsive, and socially deviant, and (2) high impulse control, heavily defended with low autonomy (Skinner et al, 1974; Nerviano, 1976). It is tempting to speculate that the features defining the first of these two groups may in part originate from the types of brain damage described earlier. Some of this damage may precede their alcoholism, for example from MBD in childhood, and interact with early socialization experiences to produce the impulsive, low ego-controlled, acting-out personality pattern. Studies relating personality data to neurological and cognitive integrity would be helpful here, as would studies relating both sets of data to a range of independent (e.g. genetic factors, early socialization) and dependent (e.g. treatment participation and outcome) variables.

Brain Damage, Cognitive Dysfunction, and Treatment Participation.

Given that neuropsychological deficits are usually associated with less effective adjustments to life tasks and demands, and given that a moderate to high percentage of alcoholics evidence these deficits throughout the period in which they are engaged in therapy programmes, the question arises as to whether the presence of this 'organicity' results in less therapeutic progress or poorer prognosis - i.e. do these person variables modify response to the environmental contingencies of both the treatment setting and the post-treatment environment?

This is a straightforward question. It is clearly a question that carries with it important implications for the treatment of alcoholism. It is also a question that has been largely ignored. Putting this another way, the substantial literature that has addressed the cognitive status of alcoholics lies largely in limbo. Little effort has been made to connect it to the day-to-day activities (some would say realities) of the clinician engaged in the difficult task of trying to get patients to modify their drinking behaviours and reorganize their lives.

Schisms between academic research and practical application are not unique to this area. To varying degrees they occur in all scientific disciplines. Closer to home, this state of affairs is seen clearly in most of the subfields of Clinical Psychology. However, it appears to be the outstanding feature of both Neuropsychology and Alcohol Studies. Further, in the latter field, the need to link the recent empirical findings with clinical practice is pressing. This urgency arises because of the limited success of conventional alcoholism treatment programmes and the apparent relevance of this area of neuropsychology to clinical issues. [1]

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1. From an extensive review of the outcome literature, Edwards (1968) concluded that only 20 percent of alcoholics have a lasting recovery. The remainder show results that vary between total failure (20 percent) and repeated relapses and remissions (60 percent). Outcomes other than recovery often involve considerable human suffering for alcoholics and those in their immediate environment.

The focus of this section is on the behaviour of patients while they are in treatment settings. The next section is oriented more towards post-treatment functioning. However, treatment activities are presumed to influence the behaviour of patients when they leave therapy. Those aspects of the individual that are not changed by the treatment experience, aspects that might include neuropsychological status, are also common to both the treatment and post-treatment situation. For these reasons, findings related to therapy can also be expected to have relevance to subsequent behaviour. It is not sufficient merely to assume this however, it needs to be demonstrated.

Although the question of the relationship of cognitive dysfunction to treatment is straightforward, there are some difficulties when it comes to finding unambiguous answers. In part this is because (1) little is known about the details of the therapy process, and (2) little is known about the relationship of most neuropsychological measures to other areas of behavioural functioning.

Given these difficulties, it is generally accepted that whatever therapy might be, and it has been claimed to be a vast number of things, it is clearly some form of learning process. The majority of alcoholism treatment programmes involve highly verbal-symbolic interchanges. Typically this occurs within a framework of group and individual therapy. Commonly, this is combined with a

more formal presentation of information about alcoholism and human behaviour, often in a lecture format. The goals of treatment usually include abstinence and the development of insight. 'Insight' can probably be regarded as the degree to which patients demonstrate a verbal acceptance of the traditional alcoholism model, particularly the need to abstain, and knowledge of the realistic changes that they will have to make in their personality and social patterns to achieve this objective. The'depth' at which they recognize or appreciate this information is also considered to be important - i.e. it is not sufficient to parrot slogans that they have heard. Having aquired this change in verbal behaviour and attitude, it is then assumed that it will generalize to behavioural changes outside of the treatment setting.

From the foregoing, it is evident that treatment progress is a complex process, or more correctly, group of processes. How can it be measured? Possibly the simplest index, and one that has often been addressed in the psychotherapy literature, is whether or not the patient remains in therapy. Other measures that have been used include: staff and self ratings of insight or more specific attitude or behaviour changes, tests of specific content aquisition, standardized tests of personality and/or psychopathology, and ratings of therapy participation.

Performance on the types of measures listed above

have rarely been considered in relation to cognitive dysfunction among alcoholics in treatment programmes. Of the few relevant studies, one compared the RFT performance of alcoholics who remained in treatment with the performance of alcoholics who left against medical advice. It was found that dropouts were significantly more field dependent than those who stayed (Karp et al, 1970). Goldstein (1976) speculated that this occurred because field dependent alcoholics lack the intellectual resources to benefit from highly verbal, intellectually demanding forms of treatment. Certainly, there is evidence suggesting that field independent individuals have greater skill in cognitive analysis and structuring (Witkin & Goodenough, 1977).

Although field dependent persons possess characteristics that could be expected to impede therapy progress, they have other characteristics that might be an asset, particularly if the therapy format was tailored to these qualities. Goldstein (1976) claims that studies relating RFT performance to behaviour in natural settings are sparse. It is evident however from Witkin and Goodenoughs' (1977) review, that this is not the case. To the contrary, it appears that this is the only neuropsychological measure that has not only been related to other areas of personality functioning, but has also been studied in relation to a wide range of interpersonal situations. This literature suggests that field dependent people are more attentive to social cues than are field independent people. They are also more interested in

other people, more emotionally open, gravitate towards social situations, more adept in important social skills, and get along better with others. Field dependent people are also more suggestible than field independent people (Linton & Graham, 1959). Presumably, these traits could be adaptive in certain situations and might be capitalized on in therapy. A further point that arises from these findings is that more attention may need to be given to ways that field dependent patients can satisfy their strong social needs while at the same time avoiding pressures to drink - pressures that could be expected in their post-hospitalization peer group. Before we can legitimately make extrapolations from this literature however, it needs to be shown that the findings derived from studies of field dependent individuals in general, also apply to field dependent alcoholics.

Apart from Karp et als' study cited above, performance on other neuropsychological tests does not appear to have been related to premature termination of alcoholism treatment. Studies have however been conducted using measures of personality, psychopathology, and demographic characteristics (Baekeland & Lundwall, 1975). Studies employing the first two categories of measures have yielded contradictory results (McWilliams & Brown, 1977). Some consistency has however been shown between other indices and dropping out of treatment. Baekeland and Lundwall (1975) summarized their review in the following way. "In sum, the composite picture of the alcoholic outpatient who is most likely to drop out of treatment

is that of the field dependent, counter-dependent, highly symptomatic, socially isolated lower class person of poor social stability who is highly ambivalent about treatment and has psychopathic features." (p. 751)

There is some consistency between the characteristics of alcoholics who prematurely leave therapy and the characteristics that have been identified among individuals in other patient groups who leave treatment at an early stage. Reviews of this more general literature indicate that those who remain in therapy tend to be of higher socio-economic status, more intelligent, more anxious, more self-dissatisfied, more willing to explore problems, more persistent and dependable, and less likely to have a history of antisocial acts (Garfield, 1971).

It is important to note that even in studies where a number of these variables have been included and evaluated by multivariate statistical methods, only a minority of the dependent variable variance is accounted for. How much of the additional variance could be taken up by neuropsychological integrity in various patient groups, including alcoholics, remains uncertain. The necessary research has not been conducted. Because of the presence of characteristics such as poor social stability, intelligence, persistence, and history of antisocial acts, indices that could show some correlation with cognitive impairments, it is conceivable that some of the independent variable variance would be shared by measures of cognitive dysfunction. Consequently,

some of the dependent variable variance that might be potentially explicable by measures of neurological or cognitive dysfunction, may have already been identified. For the present however, we can only speculate on such matters.

It is also important to recognize that both treatment type and treatment populations vary. Although there appears to be some stability in predictive characteristics across studies, the particular group of significant predictors, and certainly their importance relative to each other, could be expected to depend in part upon both of these factors. Little is known on this issue. One reason for this is because treatment modalities in the relevant studies have been inadequately described. Another reason is that many programmes appear to have been 'analytically oriented'. Although there is probably some truth in Garfield's quip that this term covers a multitude of sins, it does seem that in the main there has been considerable homogeneity in the treatment types sampled (Garfield, 1971). It could be that if therapy was oriented away from a highly verbal, insight-oriented approach, then other variables might be found to be important predictors of length of stay and treatment participation more generally.

Very little indeed is known of the relationship of brain damage and neuropsychological impairment among alcoholics to other indices of treatment participation

or progress.

From consultancy experience to a hospital ward established to attempt the rehabilitation of alcoholic Korsakoff patients, the author can attest to the painfully slow progress that is characteristic of even the most psychometrically intact patients. Horvath (1975) has made a similar observation with respect to a population of chronically brain damaged alcoholics, a population including both Korsakoffs and Alcoholic Dementias. Of 100 patients, 40 required admission to long-stay institutions. Only 14 showed significant improvement. Four from the total 100 improved significantly to achieve voluntary abstinence. A number of treatment centres are reluctant to admit patients with clinically obvious brain pathology - in recognition of their poor prognosis.

Although it is fairly certain that brain damage and cognitive deterioration sufficient to warrant the chronic brain syndrome label disrupts therapeutic progress, the evidence is thin towards the less impaired end of the spectrum. However, given the particular nature of the deficits observed in this group (see pp. 131 - 133), it could be expected that even relatively minor deficits will compromise treatment involvement and therapeutic gain.

Based on the findings of some of the experimental studies of cognitive dysfunction among alcoholics, a few researchers have suggested that demands to learn

novel complex material, or to engage in highly verbal-symbolic therapuetic regimes, should not be made on patients until at least one to two weeks after detoxification. However, the deficits that were identified and which provided the basis for these recommendations, in many instances, persist throughout the entire treatment period. Might it not be that most of these mildly to moderately impaired patients achieve little at any point in therapy? Could it not be that the very poor success rates of therapy programmes stem from inordinately high failure rates within this category of patients, rather than from failure spread randomly within treatment populations?

The second of the two questions posed in the last paragraph is addressed in the next section. Regarding the first question, there appears to be only one study in the literature, Goldman and Rosenbaum (1977), that is relevant. Other aspects of this report were described earlier. The third of three experiments outlined in their paper was designed to assess directly the ability of alcoholics to acquire novel complex information - namely information about alcohol abuse that was routinely presented as part of an ongoing treatment programme. Four different lectures were administered and subjects were required to complete tests assessing their knowledge of the material before, immediately after, and 24 hours after each lecture. No significant increases took place from pre- to post-testing. In addition, this procedure

was repeated on the fourth, fifteenth, and twenty-fifth day of treatment. No improvements occurred in learning ability over this time period. In other words, within this group of alcoholics, a group shown to be moderately impaired on other neuropsychological tests, lecture material was not learned at any point during a 28-day programme.

Unfortunately, Goldman and Rosenbaums' finding has diminished relevance for the question originally posed because it failed to subdivide the groups according to degree of cognitive dysfunction. Had this been done, it would have been possible to determine whether the overall performance was pulled down by the most impaired subjects. This experiment raises the important question of just how much learning occurs in other areas of treatment. It also questions the validity of this treatment approach.

From some of the literature discussed in chapter two, there is the further possibility that even if verbal knowledge is aquired and attitudes altered, patients with frontal damage may still have trouble translating this knowledge into action because of a lack of integration between verbal intentions and ongoing behaviour. This possibility has not been assessed.

The point just made touches on another issue that has been raised in relation to the treatment of alcoholics.

With respect to the management of patients, clinicians, like other members of society, typically make judgements about ability on the basis of verbal skills. Because verbal ability and crystallized intelligence generally, are in the main preserved, alcoholics appear to be cognitively intact. Their deficits in the area of fluid intelligence or neuropsychological integrity are often not obvious to either the patient or to treatment staff, even though they can be expected to have profound implications for attempts to alter past behaviour patterns. Consequently, alcoholics are likely to receive unrealistic assessments of their adaptive ability based upon their verbal skills (or 'verbal facade') while in structured treatment settings. This misdiagnosis of their cognitive state could pose a major problem for long-term adjustment as failure to fulfill personal expectations appears to be an important factor in relapse.

In summary, it must be concluded that much of the content of this section has been speculation - albeit informed speculation based upon what is known of the cognitive capacities of alcoholics during the treatment period. Severe neurological and neuropsychological deficits are known to severely compromise attempts at rehabilitation. Given the nature of the less severe deficits characteristic of many alcoholics, when they were considered in relation to aspects of the therapy process, it was inferred that cognitive dysfunction could be expected to affect therapy progress. However, only two studies could be located that provide some corroboration

for the view that lesser degrees of impairment might limit therapy perseverance and therapeutic change. This possibility requires fuller investigation. So too does the possibility that special programmes might be designed that take account of cognitive impairments that are typical of many alcoholics.

The Relation of Brain Damage and Cognitive Dysfunction to Treatment Outcome.

In chapter one and at other points throughout this thesis, it has been stated that clinicians and researchers have tended to see alcoholism as a unitary entity. This view has affected the design of treatment programmes. It has also had a major effect on the way treatment outcomes have been evaluated. In spite of repeated demonstrations that hospitalized alcoholics are a very heterogenous group (e.g. Wanberg et al , 1977) and that a number of intra-group differences have implications for treatment outcome (e.g. Bateman & Peterson, 1971), investigators have typically not taken subject variables into account in the evaluation of treatment outcomes. Where they have, measures of cognitive dysfunction have seldom been included and efforts have rarely been made to determine the relative importance of different factors.

The implications of the criticisms made above are profound. Apart from knowing little of the relation between neuropsychological impairment and outcome, we

also lack information regarding the major subject dimensions which need to be matched or statistically controlled in the evaluation of treatment programmes. As a result, outcome differences from one programme to another are confounded to an unknown degree by subject characteristics.

In the last section, it was noted that alcoholic patients with chronic brain syndromes show little response to therapy. Given the particular nature of the cognitive impairment typical of many less deteriorated alcoholics, it has been suggested that this category of alcoholics might also show minimal change during therapy relative to other alcoholics and, as a consequence, carry a worse prognosis. This hypothesis is in part based on the assumption that therapy has some effect on nonbrain-damaged or noncognitively impaired patients. From the evidence to date, it appears that therapy is in fact only a relatively small contributer to outcome (e.g. Armor et al, 1976). However, this conclusion arises from studies that have been claimed to have employed inappropriate statistical techniques (Cronkite & Moos, 1978). The more sophisticated use of multivariate procedures in this latter investigation suggested that individual differences, symptoms at intake, and treatment, may each account for similar percentages of outcome variance. It is not known whether the relative importance of treatment effects are greater among noncognitively impaired patients.

Apart from indirect effects on outcome by modifying response to therapy, cognitive dysfunction could also be expected to have a direct effect on post-treatment behaviour, assuming that it persists after patients leave treatment settings. Unfortunately, little is known on this issue, and the findings of the few relevant studies reviewed in chapter two were somewhat equivocal. Once expatients recommence drinking, then further cognitive impairment will arise through an interaction between chronic and acute effects of alcohol abuse.

It has previously been mentioned that with the exception of RFT field dependence, little empirical information has been gathered relating performance on neuropsychological tests to behaviour in natural settings. The only relevant data, other than case studies of individual patients in the clinical neuropsychology literature, appears to be some unpublished work by Halstead, referred to in Goldstein's (1976) review. Halstead investigated the relationship between neuropsychological test performance and supervisors' ratings of subjects' performance in work setting. As in the test situation, it appeared that more impaired individuals coped well with routinized verbal and mechanical tasks but performed poorly on tasks of a complex nature that involved conceptualization and problem-solving ability. This finding suggests that alcoholics with similar cognitive deficits might have difficulty in coping with complex demands and changes in the real world.

Rosenbaum (Note 3), in an unpublished report, outlines a model based on neuropsychological findings and related research, to explain some of the characteristics commonly attributed to alcoholics. He postulates that chronic alcoholics suffer a reduction in their behavioural repertoires and adjustive capabilities resulting from: "(a) the primary neuropsychological effects of brain dysfunction, and (b) the secondary effects of aquired aversions to social situations requiring personal efficiency" (p. 1).

Rosenbaum argues that this results in what he terms the 'alcoholic paradox', where alcoholics use overlearned routines to avoid social and job situations which demand new adaptive responses that they are unable to make and afraid to try. The inability is considered to arise because of the neuropsychological impairments that have been discussed at length above. That is to say, alcoholics may be expected to persist in well established, self-injurious alcohol-related behaviour patterns because newer response systems are "neuropsychologically less available and more difficult to acquire". (p. 4)

The fear component of the 'alcoholic paradox' is considered to reinforce the stereotypy and rigidity. It is claimed to develop from anxiety and avoidance learning that occurs when the impaired drinker experiences failure and fear of failure in situations requiring new

skills or responses. This type of secondary emotional reaction is well documented in brain damaged nonalcoholics (e.g. Goldstein, 1942). Goldstein refers to this as the'catastrophic fear reaction', a phenomenon familiar to clinicians who have tested organically impaired patients on demanding novel tasks. The author has encountered this a number of times during the administration of the Walton Black Modified Word Learning Test, where task failure is obvious to the testee.

Goldstein (1942) discusses ways in which brain damaged patients often go to considerable lengths to avoid situations and social occasions where such reactions could be expected to occur. As a consequence, organic patients typically attempt to sustain a controlled environment that is familiar and does not require novel responses. Because this learned fear reaction appears to become linked with a wide variety of environmental stimuli, Rosenbaum argues that in the case of the alcoholic, avoidance of difficult work situations, stereotyped verbal habits, and persistence in habitual behaviour patterns (e.g. visiting familiar alcohol-related situations), can be explained by this type of aversive conditioning and instrumental avoidance learning.

Rosenbaum's framework provides a positive feedback model whereby increased neuropsychological deterioration makes the alcoholic more prone to aversive conditioning and instrumental avoidance, accompanied by response stereotypy. This in turn increases the probability of

continued drinking. Sustained drinking and related self neglect in turn produces further neurological and cognitive impairment, and so on....

Rosenbaum appears to overgeneralize his model to include all alcoholics. From the last paragraph however, it is evident that the framework is most descriptive of cognitively impaired individuals. Although as yet untested in its detail, this conceptualization does provide an explanatory mechanism from which we would predict an increased probability of relapse from more cognitively impaired alcoholics.

What evidence is there that mild to moderate cognitive impairment and organic brain damage among chronic alcoholics predicts a poor post-treatment outcome?

Mann (1973) found that ventricular enlargement was closely correlated with outcome in a group of patients with cerebral atrophy. Comparison was not however made with nonimpaired patients. This finding suggests that within alcoholic populations showing neurological evidence of mild to moderate brain pathology, degree of organic deterioration is related to poor prognosis. Further research is necessary to replicate this finding and to establish whether more extensive atrophy in certain loci is more strongly predictive of outcome than others (e.g. frontal damage compared with other patterns of cortical atrophy). Inclusion of a wider range of outcome

measures would also be helpful.

A little more work has focussed on the relation of neuropsychological performance to treatment outcome. Contrary to predictions made in this chapter, Goldstein (1970) failed to demonstrate a relationship between levels of neuropsychological functioning during therapy (trichotomized into normal, intermediate, and severe) and ratings of life adjustment one year after discharge. In this study, subject size was relatively small (n = 53) and one of two alternative treatments was somewhat unorthodox, at least for cognitively impaired patients. The modality in question involved the administration of LSD. Unfortunately, the author has been unable to obtain a copy of this unpublished paper. From accounts of it in the literature (e.g. Parsons, 1977), some important details have not been made explicit. For example, when were the neuropsychological tests administered - how long after admission and when, in relation to the LSD treatments? It is also not clear what percentage of the original sample were contacted at followup. This could be important in interpreting the results as attrition is known to be nonrandom. It is not evident whether Goldstein included measures of drinking outcome as opposed to "life adjustment". There is research strongly suggesting that adjustment in other life areas bears little relation to measures of alcohol consumption, including abstinence (e.g. Gerard et al, 1962; Pattison et al, 1969; Thomas et al, 1959). Athough, from discussions above, it could be expected

that cognitive impairment would have an effect on "life adjustment". However, these discussions did not suggest that this would necessarily be the case in all life areas, only in those requiring new behaviours and more adaptive responses to avoided situations. It is even conceivable, and indeed predictable from Rosenbaum's model, that impaired patients who show some progress in these latter areas might suffer increased overt psychological distress and apparently poor adjustment in other areas (e.g. marital relationships) - at least in the shortrun. For these reasons, it is not clear what conclusions to draw from accounts of Goldstein's study.

McLachlan and Levinson (1976), using a block task shown in other studies to be sensitive to cognitive dysfunction in alcoholics, found no difference in performance between alcoholics who were abstinent one year after leaving treatment and those who were drinking. Berglund (1977), in contrast, found that alcoholics who improved their drinking habits were less impaired cognitively during treatment than those who were unimpaired. This association was not strong however. Significant differences did not exist between these two groups on any of the six individual tests employed, only on a composite, and one suspects, post hoc overall index. Followup intervals in this study ranged from approximately 2.5 to five years.

The only work that unequivocally corroborates the proposed association between cognitive functioning and subsequent outcome was conducted in our Department at the University of Canterbury (Gregson & Taylor, 1977). This study involved 90 male alcoholics with a follow-up interval of six months after they left a three month inpatient hospital treatment programme. Cognitive dysfunction was quantified by the Patterned Cognitive Impairment Test (PCIT) (Gregson & Taylor, 1974; Gregson & Abbott, 1979). This test battery was designed specifically to detect the subtle types of cognitive dysfunction that characterize chronic alcoholics. Tasks include short-term memory for the content and order of arrays of symbols, and a modification of Elithorn's perceptual mazes. The overall score is a composite of 16 separate indices, weighted on the basis of a multiple discriminant analysis that showed maximal separation between alcoholics and normal controls. Although cross validation with other more commonly used neuropsychological batteries is only now underway, extensive normative data exist from both normal and a range of clinical groups. An interesting feature of the PCIT is its negligible correlation with vocabulary knowledge, commonly considered the best measure of crystallized or 'psychometric' intelligence, and ratings of depression.

In the Gregson and Taylor study, testing took place one month after admission to the programme. The PCIT score range showed a larger spread than that obtained in other populations. Some subjects had scores more

typical of alcoholics with chronic brain syndromes (both Alcoholic Dementia and Korsakoff's Syndrome). Others had scores that were characteristic of young, well functioning normals. Many of the alcoholics obtained scores intermediate between those of normals and deteriorated brain syndrome patients.

From contingency analyses, with a three-way division of PCIT performance into high, medium, and low, it was found that degree of dysfunction significantly predicted both abstinence one month after release from hospital, and whether or not expatients held a job five months after ($p < .001$). The study also included a number of social and demographic measures that previous research had either shown or suggested bore a relation to outcome following treatment. The complete set of independent predictor variables that were employed in a stepwise linear regression analysis included the following: PCIT scores (range -6·7 to +2·0), age, social status, number of previous admissions for an alcoholism-related diagnosis, number of weeks resident at the last address before admission, marital status, A.A. membership, number of religious observances per year (self-estimated), and number of different jobs held in the last two years. The dependent variable was the number of days of abstinence up until the first relapse. Records extended to the first six post-hospitalization months.

The regression analysis indicated that of the measures

sampling behaviour prior to discharge, PCIT score was the dominant predictor variable, accounting for over 20 percent of the total variance. The probability level associated with this relationship was <.0001. When post-treatment AA membership was included in the regression analysis, this measure emerged as the dominant predictor. PCIT came second, still accounting for a large percentage of the outcome variance (17·3 percent, $p < .0001$). Of the independent variables in the set of predictors, only two others bore a relation to outcome significant beyond the ·05 level, namely social status and religious attendances. From this pattern of results, Gregson and Taylor concluded that patients with low PCIT scores, no religious affiliation, and who did not attend AA, carried the worse prognosis. A further finding of interest was that those with previous religious affiliations were more likely subsequently to join AA.

Gregson and Taylors' study is important because it not only suggests that cognitive dysfunction during treatment is a strong predictor of posthospitalization work adjustment and drinking outcome, it further suggests that it might be one of the most powerful of the various factors that influence long-term treatment outcome. It would be helpful if other prognostically relevant independent variables could be considered in relation to measures of cognitive dysfunction.

In summary, from the nature of neuropsychological

impairment typical of many alcoholics, there is good reason to expect that neuropsycholgical deficits will compromise long-term treatment outcome. There are also theoretical frameworks incorporating these and other research findings that lead to similar predictions. From these considerations, the next logical step would be to relate neuropsychological performance to measures of post-treatment processes that these frameworks and the literature on situational precipitants of relapse (see Marlatt, 1977) suggest are important. Essentially, this is a proposal for a state-trait interactionist approach to the question of post-treatment functioning. This work could be expected to furnish findings that would be valuable in the design of future treatment programmes - programmes tailored both to the cognitive state of the individual and to individually relevant post-treatment hazards.

The suggestions made above are in a sense premature. It is a case of conceptual enthusiasm outstripping empirical reality. For the moment, the results of studies linking cognitive dysfunction to treatment outcome are equivocal. We do not know with any degree of certainty that such a relationship actually exists, let alone the more intricate details of how the hypothetical situation arises.

Conclusions.

Neuropsychological investigations have been more

successful than has personality research in describing common psychological characteristics of alcoholics. However, although there are neuropsychological deficits that are quite widespread among hospitalized alcoholics, generalizations derived from this area of research do not pertain to all alcoholics. In this area of functioning as in others, alcoholics are a heterogeneous population with different types of cognitive impairment of varying degrees of severity and permanence.

Because some degree of cognitive dysfunction may predate the onset of alcoholism, it has been suggested that for some alcoholics, this may be a factor in the etiology of their problem drinking. Irrespective of its origin however, the presence of cognitive dysfunction could be expected to have important implications for the understanding of alcoholism, particularly in relation to treatment and longterm prognosis. More generally, it was postulated that some characteristics of alcoholics that have traditionally been regarded as features of personality, may in large part be a consequence of frontal lobe dysfunction. Some of the commonly observed features of alcoholics, for example, their apparent inability to abstain or to benefit from past experience, may also be due to this type of damage rather than to some intrinsic property of the 'alcoholic personality' such as oral dependency or a death wish.

The invulnerability of overlearned verbal skills could lead both alcoholics themselves, and clinicians,

to overlook the presence of other subtle, yet crucially important cognitive deficits. Given the nature of the cognitive deficits in question, this inaccurate diagnosis could lead to the development of false expectations for future adaptive capabilities. As a consequence, important areas seem to be overlooked in therapy. Additionally, in the post-treatment environment, unrealistic expectations could lead to failure in life adjustments that in turn increase the likelihood of resumed drinking.

There is a little research suggesting that cognitive dysfunction might impede response to therapy, particularly insight modes where alcoholics require intact conceptual or abstracting capabilities to acquire and utilize cognitive strategies to modify life styles and control drinking behaviour. There is also some research suggesting that the presence of cognitive dysfunction during therapy carries with it a poor post-treatment prognosis. In the main however, these areas have been little investigated and remain poorly understood. Work here has been hindered by inadequate information regarding therapy processes and the way neuropsychological deficits manifest themselves outside of the test setting. Further work is needed in these areas if programmes are to be designed that are optimally tuned to the cognitive state and other prognostically relevant dimensions of subgroups of alcoholics.

CHAPTER FOUR

OTHER MAJOR PSYCHOLOGICAL FACTORS

Introduction.

This thesis is primarily concerned with establishing what relationship, if any, exists between measures of cognitive dysfunction and both therapy participation and treatment outcome. A secondary concern lies in the identification of other psychological factors that might influence these processes and how these factors might themselves relate to cognitive impairment. The identification of further prognostically relevant psychological dimensions should, in the long-run, assist in the understanding of different individual responses to therapy. Ultimately, it might be expected that this body of knowledge would provide a solid data base from which to identify subpopulations of alcohol-dependent individuals who might be expected to profit most from different treatment approaches. Implicit in this goal is the view that unlike traditional assumptions, there is not just one population of alcoholics who can be treated by one best method.

To date, the major groups of subject variables investigated for their capacity to predict treatment participation and outcome have included: background characteristics (e.g. age, socioeconomic status, marital status), personality indices and psychiatric symptoms at intake or later in therapy, and previous alcohol

consumption patterns (both long-term and acute). Typically, multivariate investigations which have incorporated these indices into regression models to predict outcome, have only accounted for 10 to 30 percent of the variance in treatment outcome (see Bromet et al, 1977). Additional variance (0 - 20 percent) is often explained when treatment type and/or treatment participation are added. From Bromet et als' (1977) and Armor et als' (1976) reviews, it appears that all of these indices combined can 'account for' between 15 to 50 percent of the variance in treatment outcome, the total depending upon the population sampled and the outcome measure employed. This leaves 50 to 85 percent unexplained! Bromet et al consider that the most important question for future research is to identify what factors 'explain' the remaining variance in treatment outcome.

Apart from Gregson and Taylors' (1977) study cited in the last chapter, measures of cognitive dysfunction have not been evaluated in relation to the varied indices described in the last paragraph. In their study, cognitive dysfunction alone accounted for 20 percent of the dependent variable variance - more than that accounted for by the entire range of predictors in some studies. The further finding of this study was that post-treatment participation in Alcoholics Anonymous accounted for another 20 percent of outcome variance. Although it is necessary to attempt to replicate these results using new samples and additional outcome measures, this work suggests that two powerful

and yet currently ignored groups of 'explanatory' predictors include neuropsychological functioning during treatment and measures that pertain to the post-treatment environment.

In the main, measures of psychological functioning during treatment have not been very useful in predicting outcome. As indicated, cognitive indices have not typically been employed. Measures of psychiatric symptomatology on the other hand have been used. In chapter three, it was mentioned that depression and psychopathy are typical of many alcoholic patients in treatment settings. A few studies have found that the greater the depression or sociopathy, the more likely the treatment failure (e.g. Baekeland et al, 1971; Bowman et al, 1951; Lundwall & Baekeland, 1971). Other studies have failed to establish this relationship (see McWilliams & Brown, 1977; Neuringer & Clopton, 1976 for reviews).

Personality measures have often been used in studies attempting to predict those who leave treatment prematurely as opposed to those who stay. In both this task, and in attempts to predict other treatment changes (e.g. 'adjustment'ratings), personality factors have generally failed as significant predictors (e.g. Gross & Nerviano, 1973; Hoffman & Jansen, 1973; Pryer & Distefano, 1970; Wilkinson et al, 1971). Personality change scores over the course of therapy have also failed in this task (e.g. McWilliams & Brown, 1977).

A few studies have used objective personality test scores and score changes during treatment to predict drinking behaviour at varying times after treatment. As with attempts to predict changes during therapy, personality variables have proved to be of little value in this respect (e.g. Trice et al, 1979; Muzekari, 1965; Sikes et al, 1965).

In spite of poor success in the research efforts to link personality functioning to treatment participation and post-treatment outcome, Neuringer and Clopton (1976) conclude their review by stating, "There is a clear need for more research looking for relationships between objective personality test scores and the outcome of treatment for alcoholism". (p. 19) Later they add, "Given the diverse personality patterns found among alcoholics, two related questions are suggested for future research. First, are alcoholic patients of a certain personality type more likely to profit from alcoholism treatment programmes than alcoholics with other personality patterns? Second, can an alcoholic's personality test scores be used to indicate which type of treatment will be of most benefit to him?". (p. 20)

From a consideration of the studies upon which Neuringer and Goldstein base their conclusions, the present writer is more inclined to consider that in terms of augmenting predictive models, this area may have already been sucked dry. Metaphorically speaking, the enterprise may be one of attempting to draw blood

from a stone. Given the promising lead of Gregson and Taylors' (1977) investigation, might we not be better employed reorienting our energies toward researching the potential that may exist in the areas of cognitive functioning and post-treatment experiences?

Which processes or traits of the individual are considered to be attributes of cognition as opposed to personality varies from one writer to another. The division is somewhat arbitrary. Further, in everyday activities, aspects of both interact in complex ways to produce behavioural outcomes. Earlier, it was mentioned that even performance on carefully structured tasks such as neuropsychological tests, is a result of factors other than just the organic state of the brain and/or cognitive processes. For example, attention, emotional state, and motivation, are important factors to consider.

In chapter two, intelligence was defined in a way that included elements typically regarded as belonging in the domain of personality. The converse is also common, with intelligence and cognition incorporated into definitions of personality. For example, Griffiths (1970), defines personality as "the more or less stable organization of a person's emotional, conative, intellectual and conceptual, and physiological behaviour which determines to a large extent his adjustments to environmental situations". (p. 83)

"Intellectual" as used by Griffiths, refers to the form and structure of cognitive behaviour. This aspect of cognition among alcoholics has been discussed in this thesis under the headings of intelligence, cognitive dysfunction, and neuropsychological impairment. Emphasis in these areas is placed upon the way in which different types of information are processed and include: perception, storage, manipulation, integration, and retrieval.

"Conceptual", as used in Griffiths' description, denotes the content of cognition - the "what" rather than the "how" of cognitive processing. Included under the "whats" of cognition are specific knowledge, attitudes (which also include an emotional component), and personal constructs - including both specific and more general schemas.

The conceptual aspects of cognition take a central position in Kelly's Psychology of Personal Constructs (1955), as well as forming the main datum for a number of cognitive and cognitively oriented behavioural theorists (e.g. Rotter, 1966). This aspect of the individual's psychological functioning has been little investigated in relation to therapy participation and outcome among alcoholics. Additionally, it has not been included in recent major reviews of either the cognitive or personality literatures pertaining to alcoholism.

Although little studied, there is some work suggesting that this subfield of the cognitive literature may have more to offer in the furthering of our knowledge of therapy-related issues than those areas more commonly addressed by personality theorists.

This chapter reviews a number of areas within the 'conceptual' area of cognition, as they apply to alcoholism. These reviews, in the main, are not as exhaustive as those in previous chapters. In large part, this is because relevant studies are either lacking or widely scattered in the literature. Unlike earlier chapters, this one is somewhat piecemeal. Each area discussed stands largely on its own. This occurs because there is no integrating model or theory which is capable of adequately linking the various concepts and measures that operationalize them.

Locus of Control.

Introduction.

In 1966, Rotter, building on earlier work by Phares (1957) and James (1957), developed the concept of locus of control of reinforcement. At this time, he also developed a questionnaire to operationalize his construct, the I-E Scale (Rotter, 1966). Since then, vast numbers of studies have been conducted using the I-E Scale and related measures of control orientation. Levenson and Miller (1976) claim that there are over 1000 published

studies. These studies come from many of the subfields of psychology. It is only during this decade, however, that locus of control has been studied in relation to alcoholism.

Locus of control (abbreviated from the earlier locus of control of reinforcement) refers to individuals' generalized expectancies about whether or not they have control or power over what happens to them. It is conceptualized as a continuum along which individuals are normally distributed. One end is termed internal control, defined as "..the perception of positive and/or negative events being a consequence of one's own action and thereby under personal control". The other end is termed external control - "..the perception of positive and/or negative events being unrelated to one's own behaviours in certain situations and therefore being beyond personal control" (Rotter et al, 1972, p. 499). Rotter claims that in Western cultures, external causation is attributed to either chance, luck, fate, or to powerful others, or to the complexity and hence, unpredictability, of the environment.

Abbott (1977) reviewed the general locus of control literature with a view to building a state-trait model of helplessness by incorporating elements from a variety of conceptual frameworks including: locus of control, learned helplessness, reactance theory, and attribution theory. From the review of the locus of control research, it was concluded that externals do perceive reinforcements

to be less contingent upon their own behaviour than is the case with internals. Further, locus of control orientations have been shown to be fairly stable and to exert an influence upon a wide range of behaviours in a variety of situations.

Consistent with their increased expectancies of control, internals tend to have higher achievement motivation, show more attempts to influence their environment and realize goals, and to be more persistent at skill tasks. These characteristics are also reflected in their positive self images.

Externals differ from internals in the above respects. Consistent with their experiences of powerlessness and frustration in relation to external events, they display more anxiety, hostility, aggression, and mistrust of people. Their childhoods are characterized by unpredictability and punitiveness. They are over-represented among lower socioeconomic classes, ethnic minorities, and women.

Failure experiences are typically retrospectively devalued by externals and attributed to environmental causes. Internals, in contrast, are more likely to attribute failure to either their own lack of effort or to their incompetence. Unlike the externals' sensitization defences, they more commonly cope with anxiety arising from failure by repression.

Externality has also been found to be related to enduring symptoms of depression and other depression-related phenomena.

The summary of the locus of control literature outlined above is oversimplified. This is unavoidable when a phenomenon is complex and when the relevant literature is vast, and in places ambiguous. Two qualifications must be made. First, most research in this area, and the summary just outlined, presents locus of control as a typology. Originally, it was proposed as a continuum and, indeed, the majority of individuals in populations studied, have been found to be centrally distributed. Second, there appears to be a subgroup of externals ('defensive externals') who display the external's defensive mode, and yet act like internals in many other ways.

Measures of Locus of Control.

A large number of different measures of locus of control now exist. A number of these have been used with alcoholics. The most widely used in both the more general literature and in that pertaining to alcoholics is the original I-E Scale. This scale is a 29-item, forced choice questionnaire which includes six filler items to increase the ambiguity of the test's purpose. The items sample a wide range of situations in which I-E attitudes are expected to affect behaviour. Rotter's

(1966) account indicates that the test has consistent and acceptable test-retest and internal consistency reliability. Good discriminant validity is reported, showing very low correlations with intelligence, social desirability, and political affiliation. Construct and criterion-related validity were evidenced by the demonstration of predicted relationships between locus of control and both expectancy changes on a laboratory task and active efforts by tubercular patients to improve their condition. Two factor analyses were reported. Both identified one general factor accounting for the majority of the variance.

Subsequent research with the I-E Scale has supported the early reliability claims and confirmed the low correlation with intelligence. Social desirability correlations have been more variable. Like all questionnaires, it appears that this scale is subject to error in particular test situations. Recent studies have also found a more complex factor structure (Collins, 1974; Gurin et al, 1969; Lao, 1970; Mirels, 1970). There are indications that these differences are due in part to changes in social attitudes since the scale was first developed and that these changes are also reflected in the change in the mean university student score from eight (SD = 4) to between 10 and 12 (Rotter, 1975). This represents a trend over time in the external direction and it appears that the increased complexity of the scale reflects an increasing differentiation in attitudes, particularly among individuals

scoring at the external end. In defence of Rotter's claim, that the I-E Scale is a measure of generalized control expectancies, is the finding that the additional factors appear to show instability across populations.

Other scales of locus of control are described elsewhere (e.g. Abbott, 1977; Rohsenow & O'Leary, 1978). Some of these tests are tailored to more specific areas, for example, expectancies of control over scholastic outcomes. In the next section, a scale will be described that was developed to measure expectancies relating to drinking and alcohol-related behaviours. Levenson (1973, 1974) has constructed separate scales for expectancy of control by self, powerful others, and chance. This represents an attempt to quantify the increased complexity of the external dimension.

For general purposes, the I-E Scale still appears to be the preferred instrument. Levenson et als' test may in the long run prove more valuable if different correlates of the separate scores can be established. If more accurate predictions are sought in particular areas, then domain-specific tests will need to be constructed.

Locus of Control in Alcoholics and Controls.

The task of describing locus of control in relation to alcoholism is greatly assisted by Rohsenow and O'Learys' recent review of this literature. These writers suggest

that locus of control has recently caught the attention of researchers in the alcoholism field because of its apparent relevance to aspects of the treatment process. In particular, a major concern of psychotherapy involves helping the patient to learn how to exert control over events that affect him or her. In part, this involves assisting the patient to identify causal relationships between his behaviour and subsequent events. Thus, it is further suggested that an important goal of therapy would appear to be the development of a more internal control orientation. Rohsenow and O'Leary make the point that although this seems reasonable, especially in light of the inability that alcoholics have in controlling their drinking behaviour and in coping effectively in other life areas, it may not be so straightforward. This is because alcoholics typically believe that they can control their drinking, in spite of evidence to the contrary. If this unrealistic expectation of control is reflected in measures of internal control, then the goal of increasing internality might not be appropriate.

It was originally hypothesized that alcoholics in treatment settings would be more external than nonalcoholic controls. This hypothesis seemed reasonable given that alcoholics appear to have limited control over their drinking behaviour, as well as having difficulty coping in many other life areas. Further, in a wide variety of populations, external control has consistently been shown to be associated with the presence of a number of forms of

psychopathology whereas internality has been associated with psychological adjustment and adaptive behaviour (e.g. Joe, 1971).

A number of studies have now addressed the hypothesis posed in the previous paragraph. Early studies failed to corroborate this view. To the contrary, they suggested that rather than being external, alcoholic populations are excessively internal in control orientation. Unfortunately, these studies contained a number of methodological flaws, the major one being the inclusion of innapropriate control groups (see Butts & Chotlos, 1973). Consequently, the differences between the groups could have arisen from factors other than alcoholism, for example age, education, and social class - all factors known to influence locus of control scores.

From Rohsenow and O'Learys' (1978) review, it is evident that recent studies, using more suitable control groups, have generally found no difference between alcoholics and normals. The exceptions were instances where alcoholics were more external. There is a further suggestion from one study (Norwicki & Hopper, 1974), that female alcoholics may be more external than both male alcoholics and normal females.

Concluding this section of their review, Rohsenow and O'Leary make the following comments. "The question of whether alcoholics in general tend to be more internal or external than similar persons who are not alcoholic

is not clear". "Replications are sorely needed, using different populations of alcoholics and better controls. Control groups need to be carefully matched on any variable which may reasonably be expected to influence locus of control scores, starting with age, sex, educational level, and specific occupation". (p. 68)

Changes During Treatment.

Rohsenow and O'Leary cite three studies where significant shifts in the direction of increased internality over the course of treatment was found. They located three studies where no significant changes were observed. None of the six studies included controls for practice effects. One of these studies (Chess et al, 1971) is of additional interest because of the inclusion of other measures. Thirteen alcoholics and 13 hospital employees were tested weekly for field dependence and arousal, and bi-weekly for I-E. Alcoholics became significantly more internal from testing to testing over a seven week assessment period (mean scores shifted from 6·0 to 3·9). Hospital employees failed to evidence significant change (2·3 to 3·2). The alcoholic group also showed a significant tendency for change toward decreased arousal, decreased field dependence, and decreased externality to occur in the same subjects.

O'Leary et al (1976) found that alcoholics who entered therapy with internal scores became more internal

over the treatment period whereas those starting with external scores did not. This raises the possibility that overall changes may in fact represent somewhat larger shifts within a subgroup of patients. It could be that significant changes in this group occurred in the studies where change was not evident. Shifts in this group could have been swamped by inertia within initially external subjects. Gender has not been considered with respect to I-E changes over time. Although not yet investigated, it might be that some of the confusion noted in relation to I-E scores in alcoholics compared with controls, arose because of differences in the stage at which tests were administered.

Relation of Locus of Control to Other Measures.

A second review article by Rohsenow and O'Leary (1978b), discusses research with alcoholics dealing with the relationship of locus of control to age, ability to function, and personality traits. Some of these studies have considered the relationship between locus of control and intellectual, social, and cognitive functioning. From this work, there is a suggestion that internality is associated with better social functioning. Regarding the other measures, the results are inconsistent and no conclusions can yet be drawn. The same goes for the relationship between age and locus of control. Field dependence (as measured by the embedded figures test), has been shown to correlate highly with a variety of neuropsychological tests (see chapter 2). It fails,

however, to correlate significantly with locus of control.

Studies relating I-E performance to indices of personality and psychopathology are somewhat more interpretable. As in nonalcoholics, externality is associated with increased helplessness, depression, anxiety, isolation, and general psychopathology on the MMPI. It appears however, that this may only be the case with males. For females, the only correlation of any significance is with anxiety. Internal and external alcoholics also seem to differ in the defence mechanisms that they typically employ. In this respect, the results parallel findings from studies with nonalcoholics.

In this area, as with other studies exploring the relation of locus of control to alcoholism, a large number of problems in both conceptualization and methodology are evident. Rohsenow and O'Leary (1978b) identify the following: (1) much of the work has been shallow, incorporating measures of locus of control without any theoretical rationale; (2) research has used biased samples, including inadequate representation of female alcoholics; (3) research has treated alcoholics as a homogeneous group when they are known to be heterogeneous - consequently, it is hardly surprising that results show large variance; and (4) studies have been based on the assumption that a linear relationship exists between locus of control and other variables when it is possible that a curvilinear relationship exists

with both extreme internality and extreme externality being maladaptive.

The Relationship of Locus of Control to Treatment Participation and Outcome.

The finding of better psychological adjustment among internal alcoholics, along with other indications that internal alcoholics share qualities characteristic of internal nonalcoholics (e.g. higher achievement motivation, more optimistic future outlook) has given rise to the hypothesis that internality may be positively related to alcoholic rehabilitative success (e.g. Rohsenow & O'Leary, 1978b). It has also been suggested that alcoholics with more internal scores should respond better to therapy because they can better govern their behaviour with respect to social reinforcement (Query, 1972).

Questions that arise from the above considerations include the following. (1) What effect does initial locus of control score have on treatment participation (TP) and treatment outcome (TO)? (2) What effect do locus of control shifts as a function of therapy have on TP and TO? (3) What effect does final locus of control score have on TP and TO?

Rohsenow and O'Leary cite only one adequately controlled study that addressed some of these questions.

O'Leary et al (1976) administered the I-E Scale at the beginning and end of an eight-week treatment programme. A condition of programme entry was that patients agree to attend weekly group therapy sessions for a year following discharge. Patients who completed this part of the treatment contract were then compared with those who dropped out. No differences were found between the two groups on initial I-E score or in change in I-E score over the treatment period. However, the second I-E score was significantly more internal for dropouts. This appears to be inconsistent with what was expected. However, the meaning of this result is unclear because it is not certain whether dropping out of outpatient aftercare occurred because patients could actually cope more effectively and considered that they needed no further treatment, or whether they unrealistically believed that they could cope.

Two more recent studies have also addressed this issue. Castor and Parsons (1977b) administered Levenson's tridimensional locus of control scale to 27 male inpatients at the end of their second and tenth weeks in treatment. A second sampleof 38 patients (gender unspecified) who were all treatment failures, were administered the Levenson Scale at the end of a two-week inpatient programme and again at the end of a six-month outpatient programme that followed from their short residential stay.

Levenson's scale provides measures of three different

aspects of control orientation: locus of control-internal (LOC-I), the extent to which the individual feels in control of the future through his or her personal action; locus of control-chance (LOC-C), the subject's view of the role chance plays in his or her life; and locus of control-powerful others (LOC-PO), the subject's view of the role others have in what happens to him or her.

On none of the three subscales was there evidence of a significant change over time. In the first sample, locus of control scores were not related to drinking outcome 12 months after treatment. In the second sample however, initial LOC-C scores were significant predictors of outcome. In contrast to the patients in the first sample, patients who were undergoing their first treatment for alcoholism, the second sample consisted of recidivists who had had a mean of two previous admissions. From this, Castor and Parsons concluded that "alcoholics who have been in several previous treatment programmes and who have strong beliefs that chance controls their lives appear to be poor therapeutic risks.." (p.2093).

An interesting feature of the Castor and Parsons report was the inclusion of measures of depression, hopelessness, and psychopathy. At the first testing, similar correlations were found between locus of control and these symptom measures in both the treatment successes and failures. At the second testing, differences

between these two groups emerged. Among the successes, attitudes of hopelessness shifted from an initial association with external control by powerful others and chance to an association with internal control. Among the failures, the initial relationships persisted at the second testing. This could be interpreted as a tendency for successful patients to take responsibility for behavioural outcomes, even though they don't consider that they have any more control over them. In other words, they recognize a cause and effect relationship between their behaviour and subsequent outcomes. Rotter (1966) considers that this perceptual set is a prerequisite for behavioural change to occur. This finding also suggests that the effect of control orientation on outcome may manifest itself in complex ways through relationships with intervening variables.

In a second study, Castor and Parsons (1977a) investigated the relationship between locus of control, depression, and sociopathy, in relation to treatment outcome. Psychological assessment took place at the end of the second week in treatment. Again they found that Levenson's LOC-C predicted outcome among male alcoholic recidivists (patients who had had a mean of three previous admissions). However the sample size was small. Twenty-three subjects were involved, 11 of whom were drinking at four to six months after discharge, 12 of whom were sober. This relationship did not hold among other groups of alcoholics.

The results of this study also suggested that although sociopathy per se did not seem to influence outcome, if linked psychologically to a high LOC-C orientation, then it was associated with treatment failure. A further finding was that among less successful patients, depression was also related to LOC-C. Among successful patients, in contrast, depression was related to LOC-PO. These findings are somewhat complex. Again, they indicate that the relationship of locus of control to outcome may be mediated through relationships with other variables.

From the studies cited above, it is obvious that very little is known of the relationship between locus of control and therapy participation and outcome. It is not even known whether internality or externality carries with it a better prognosis. From Castor and Parsons' work, it is possible that such effects may only become evident if other conditions are also present and/or when separate components of control orientation are considered. The possibility that extreme internals show poor adjustment because they believe that they are more in control than is warranted by reality has also been neglected generally, not just among alcoholics.

Rohsenow and O'Leary conclude this section of their review by stating:

> Change in individuals' locus of control scores over the course of treatment or followup should be examined in relation to abstinence,

> controlled drinking, and a return to heavy drinking within a sufficiently long follow-up period. Related variables need rigorous control since factors such as age, education and organic damage may more parsimoniously account for differences among groups. (p. 73)

Clearly, there is also a need to follow their recommendation with initial and final locus of control scores. Their comment regarding organic damage is interesting. Essentially, what they are calling for is a multivariate analysis with a range of potentially prognostic indices included alongside of measures of locus of control. A start has been made by Caster and Parsons. They considered three independent variables. However, the statistical analysis of their data was confined to t-tests and within-cell bivariate correlations.

Conclusions.

Although the locus of control construct appears to have relevance to the treatment and understanding of alcoholism and its treatment, although a large number of speculations have been made regarding possible relationships, and although over 30 studies have been conducted, very little can be concluded. Methodological problems have been the major factor in producing this unsatisfactory state of affairs.

From the confused findings discussed above, it is not even clear how relevant locus of control is to alcoholism. Although only more refined and more

adequately controlled research strategies can settle the issue, nonlinearity or multidimensionality of locus of control may mean that the relationship is more complex than linear statistical models can adequately handle. More consistent results might also emerge if alcoholic populations were subdivided according to clinically and psychologically meaningful criteria. A start would be to describe adequately the alcoholic sample used in a given study! Dividing treatment populations into males and females also seems sensible given the results of some of the studies described above and further research suggesting that among nonalcoholics, internality and externality have different antecedents in males and females (Abbott, 1977). Castor and Parsons' finding that LOC-C predicts relapse among recidivists but not among novice alcoholics suggests another division. Using the entire score range, or trichotomizing locus of control score distributions would also seem logical. If there are nonlinear relationships, the mean or median split that has been so popular will prevent the investigation of this possibility.

Drinking-Related Locus of Control.

Introduction.

The vast majority of research in the locus of control field has treated the concept as a general character trait and has been concerned with its relationship to other psychological constructs, as well as with its

capacity to predict behaviour in a wide variety of settings. Its origin however, lies within social learning theory. There, its function is somewhat different. Within this framework, one which attempts to integrate S-R and cognitive/field theories, locus of control is but one behavioural determinant - a relatively unimportant one at that (see Rotter, 1975).

Within social learning theory (Rotter et al, 1972), expectancies are conceived as being only one of three major determinants of behaviour. The other two are the value of reinforcement and the psychological situation. Additionally, locus of control is but one expectancy, a very broad one covering a wide range of situations. In any given situation, the expectancy of success and/or failure is a composite of information that the individual has from current and past experience with that situation, from experience with similar situations, and finally, from the individual's most broad expectancies about control over situations in general.

Two points follow from the above discussion. First, locus of control was not intended to be a precise predictor of behaviour in a given situation. It was intended to provide a low degree of prediction over a wide range of situations. For the former purpose, information is required on more precise expectancies. Second, it follows that the more ambiguous the situation to an individual, the less more specific expectancies will be able to operate. Consequently, generalized

expectancies will be relied upon more and, in this situation, locus of control could be expected to afford quite high predicitive capacity. As a rule in locus of control research, these considerations have been overlooked. Consequently, a degree of predictive ability has frequently been sought where locus of control per se would not be expected to provide this.

The criticism made in the last paragraph may well apply to attempts to predict drinking outcome from locus of control scores. Afterall, alcoholics have had a great deal of experience with drinking situations. It could be that more specific expectancies are of greater importance in influencing behaviour relating to alcohol use. Generalized expectancies of control may show only a weak relationship to behaviour in this area. They could however, have more relevance to other treatment outcomes.

The Drinking Related Locus of Control Scale.

For alcoholics, alcohol consumption and dealing with alcohol related experiences is hardly novel. Presumably, such situations do not involve ambiguity as far as behavioural expectancies are concerned. Consequently, as suggested in the last section, it could be expected that alcoholics' behaviour in such situations will be influenced by specific expectancies related to these situations rather than more general behavioural expectations. Following from Rotter's (1975)

suggestion that by developing measures of more specific expectancies, predictive ability can be enhanced in circumscribed areas, Keyson and Janda (Note 4) developed a drinking related locus of control scale (DRIE). This scale is considered by Oziel and Obitz (1975) to represent a translation of generalized locus of control into specific locus of control with regard to drinking behaviour.

Donovan and O'Leary, in a recent series of papers, describe work outlining the factor structure and reliability of this measure, along with further studies relating the DRIE to personality measures and drinking related variables (Note 5).

Three factors, accounting for 65·5 percent of the total scale variance have been identified. The first factor (Intrapersonal Control Factor) concerns the individual's perceived inability to resist the temptation to drink and his or her drinking to reduce negative emotional states. The second factor (Interpersonal Control Factor) seems to cover perceived inability to resist social pressures to drink and inability to cope adequately with anger or frustration-producing interpersonal situations. The third factor (General Control Factor), identified by only three items, is concerned with the belief that the individual's sobriety is maintained by chance factors.

Donovan and O'Leary consider that this scale has considerable potential as a research tool for furthering our understanding of alcoholism treatment and relapse. They note that the two main factors refer to areas where alcoholics appear to have particular difficulty - the areas of inter- and intrapersonal control. It was noted in an earlier chapter that problems in these areas appear to be the major reasons for relapse following treatment.

Donovan and O'Leary explored the concurrent validity of the ORIE. As predicted, they found significant, low order correlations with other measures of locus of control including the I-E Scale. The ORIE also evidenced generally similar correlations to the I-E Scale with a variety of personality and psychopathology measures. It showed discriminant validity by having only minuscule correlations with measures of cognitive dysfunction and the Sociopolitical Control factor (Mirels, 1970) of the I-E Scale.

From the pattern of intercorrelations, the alcoholic with an external ORIE score appears to present a clinical picture similar to that of alcoholics who are external on the I-E Scale. They see the outcome of significant life events as being beyond their control and determined largely by chance. They perceive themselves as having little control over both intra- and interpersonal sources of stress. ORIE externals are also claimed to experience more depressive symptoms, to be more self critical, and

to be more inhibited and indecisive. This group, in addition, appears to be more sociopathic and to have difficulties in interpersonal communication and in other social skills.

Donovan and O'Leary also compared the performance of alcoholic males with the performance of an appropriate nonalcoholic group on the DRIE, the I-E, and the Beck Depression Inventory (Beck, 1967). Testing of the alcoholic patients took place approximately 1·5 weeks after admission to a treatment centre. Although alcoholics and nonalcoholics did not differ significantly on the I-E, the alcoholics were significantly more external on the DRIE and significantly more depressed. The locus of control findings remained essentially the same when the effects of depression were removed by covariance analysis.

The authors cited in the last paragraph also evaluated the relationship between four locus of control groups, formed from median splits on the I-E and DRIE scales, and scores on the Alcohol Use Inventory (AUI; Horn et al, Note 6). I-E performance failed to show any main effects on AUI scores. DRIE externals however, differed significantly from DRIE internals on 10 of the 20 AUI scales. That is to say, they had higher scores on: Obsessive-Compulsive Drinking, Prior Use of Help to Stop Drinking, Loss of Control When Drinking, Social Role Maladaption, Psychoperceptual Withdrawal, Obsessive-Sustained Drinking, Alcoholic Deterioration (primary and

secondary), and General Alcoholism level.

In summary, it seems that the DRIE has relevance to a range of indices that previous research has shown to be of value in furthering our understanding of alcoholism. It also appears that this measure may provide a corrective to the difficulties more generalized measures of control orientation have faced when predictions of alcohol-related behaviour were sought. Donovan and O'Leary acknowledge that research with the DRIE is still in its infancy, and that further work on its concurrent and construct validity is needed to determine its theoretical and practical utility. Although promising, it needs to be noted that to date, the relationship of DRIE performance to treatment participation and outcome has not been investigated.

Time Perspective.

Introduction.

This section provides a cursory account of a complex phenomenon. The major concern is to describe the concept of time perspective, to outline a few studies that have investigated time perspective in alcoholic samples, and to consider the possible relevance of this concept to increasing our understanding of treatment changes and post-treatment functioning.

Time perspective has been defined in a number of different ways and clearly means somewhat different things to different investigators (see Black, 1969 for a review). Many researchers have been concerned primarily with future time perspective, defined by Wallace (1956) as "the timing and ordering of personalized future events". Other writers include past and present dimensions. For example, Einstein (1964) describes time perspective as "..man's ability to project himself into the future or the past (i.e., memory for past events)". Blatt and Quinlan (1967), refer to time perspective as "the capacity to relate current experiences to a historical past and to an anticipation of the future".

Black (1969, p. 16) quotes a summary of the time perspective concept given by Wallace and Rabin (1960). These authors state:

> Whereas the experiments in the perception and estimation of time deal with relatively brief periods of time (usually seconds and minutes), time perspective is concerned with long periods of time, the limit of which is one's experienced lifespan itself. The units of time, in addition to direction (e.g. past future etc.), are usually days, weeks, months, years, and decades. Here the time periods are not artificially limited by E's production of stimuli; the 'points' in time which delimit the periods are important events in the life of each subject. It follows therefore, that time perspective involves a molar rather than molecular (or atomistic) approach to the problem of temporal behaviour. It involves the total personality, memory for past events, and hopes, aspirations, and anticipations of future events. The data which are obtained in a'projective' fashion involve temporal

> quantity ('extension') as well as orderly arrangement of events in a logical sequence ('coherence') and direction or temporal 'orientation' (whether primarily past, present, or future).

Time perspective, particularly the ability to predict or anticipate the future, is considered to be an important feature of human psychology. Its development in adult humans is considered to qualitatively differentiate the organization of their behaviour from that of children (Allport, 1955) and subhuman animals (Mowrer & Ullmann, 1945). Arieti (1947) argues that future anticipation "occupies the greatest part of man's thoughts, and consequently determines the greatest number of man's actions".

The development of a time perspective allows the individual to build up a conceptual model of the future to which he or she relates past and present experience. This model provides a framework of long-term goals in terms of which the individual orients current thoughts and actions. Thus, a well developed time perspective may be seen as essential for purposeful, goal-directed behaviour. Consequently, time perspective may be seen functionally as the means by which individuals are removed from the constraints of their immediate concrete situation and as a mechanism which provides alternatives to impulsive behaviour linked only to stimuli of the present. More phenomenologically oriented writers (e.g. Kastenbaum, 1964), see time perspective as providing

a "..framework within which self identity develops, maintains and transforms itself".

Response inhibition, in a sense, is the other side of the coin of time perspective. Without it, impulsive behaviours triggered by immediate concerns and external stimuli would interfere with the realization of long-term goals. Without a developed time perspective, there would not be long-term goals or a means of linking past and present to the future.

Alcoholics, as a group, have often been described as unable to defer immediate gratifications (e.g. McCord & McCord, 1960).[1] They have also been characterized as being pessimistic about the future (e.g. McKay, 1961) and unable to formulate or realize long-term goals (e.g. Shefrey, 1955; Singer, 1950). Other writers, for example Gliedman (1956), have suggested from clinical observations, that alcoholics seem to live in an 'extended present' unaffected by past or future. Although poorly quantified, these observations raise the possibility that a number of alcoholics, if not the majority, have an impaired or undeveloped time perspective, particularly with regard to future orientation and extension. As the capacities described above are important in relating current behaviour to intentions for the future and, presumably, in motivating behavioural change, it could

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1. Also see earlier sections of this thesis, particularly discussions of frontal lobe damage and sociopathy.

be expected that a study of time perspective will further our understanding of alcoholism and have implications for treatment.

Time Perspective and Alcoholism.

The first empirical investigation of time perspective in an alcoholic population appears to be that of Roos and Albers'(1965). They administered the Time Reference Inventory (TRI) to 35 alcoholic inpatients (31 males and four females) and 27 control subjects who did not "manifest signs of psychiatric derangement" (five males and 22 females). The groups did not differ in age, intelligence, or educational level. Although there was a gender imbalance, a previous study by the authors using the TRI failed to show any evidence of gender differences.

Alcoholics were found to have a shorter future extension (mean = 3·5 years) than the controls (mean = 9·1 years), $p<·001$. No difference was found with regard to past extension, although alcoholics selected significantly more positive events in this category. The authors interpreted the alcoholics' truncated future extension as being compatible with the view that alcoholics are concerned primarily with short-range gratification and have difficulty in maintaining long-range goals. The over-representation of positive events

in the past was considered to suggest a "nostalgic yearning for the past as a fantasized period of happiness" - presumed to be a regressive defence.

A second study in this area was concerned only with future time perspective, defined by Wallace (1956) as "the timing and ordering of personalized future events" (Smart, 1968). Smart was concerned with two components of future time perspective: (1) extension ("the length of future time which is conceptualized"), and (2) coherence ("the degree of organization of the events in the future time span").

Thirty-three alcoholic outpatients and 33 nonalcoholics of similar age, sex, marital status, and occupational category to the alcoholics, were included in Smart's study. Two types of task taken from Wallace (1956) were used. One measured extension, the other measured both extension and coherence. Striking differences were found between alcoholics and the controls in both aspects of future orientation.

On unstructured tasks with no explicit mention of time, the alcoholics showed far less extensive future orientations. On a task which required subjects to "tell me ten events that refer to things that may happen to you during the rest of your life", followed by "and how old might you be when that (i.e. each of the 10 items in turn) happened?", alcoholics obtained a median score of five years - in contrast to a median of 22 years

for the controls.

Coherence was measured by a rank-order correlation between the ranking of events based on age of occurrence in the task just described and a subsequent ranking of the same 10 items when subjects were instructed to "arrange these cards (i.e. events) in the order in which they might occur." These tasks posed no problem for any of the social drinker controls. However, only 12 of the 33 alcoholics could complete the tasks. Smart commented, "It was surprising to find this task so difficult for nonpsychotic alcoholics, most of whom were employed or recently employed, living with their families and functioning in a minimally adequate way in many non-drinking situations." Even for the 12 alcoholics who could complete this section of the testing, their coherence scores were significantly lower than those of the social drinkers.

In this study, a significant correlation (-.44, $p < .001$) was found between age and future extension for the alcoholics. The corresponding correlation for the controls was nonsignificant.

Smart makes the following comments in his discussion of these results:

> These findings do not clearly indicate whether the shortened and distorted future time perspective results from uncontrolled drinking or whether it is a prior selective factor in the development of alcoholism. However, the high negative correlation between extension

> and age (and presumably years of drinking) suggests that it may be a coping mechanism developed during uncontrolled drinking. Perhaps as their drinking gets increasingly out of control, and as unpleasant consequences build up (e.g. difficulties with employment, family stability, and general health), the alcoholic perceives such a bleak future that he refrains from extending it or ordering what little future is perceived. An alternative explanation is that alcoholics cannot take account of the negative consequences of their drinking because to do so requires an elaborate time perspective. When the impulse to drink heavily occurs, it is readily acted upon because the lack of a future orientation prevents consideration of sanctions or other negative reinforcements contingent upon drunkeness. (p. 83)

Smart's two suggestions need not be mutually exclusive as he seems to imply. Presumably, both could apply. The alcoholic may or may not be less future oriented initially. However, once alcohol abuse is underway, this could lead to increased truncation and disorganization which in turn reduces impulse control with regard to drinking behaviour. Once again, we have a feedback model which could be important in explaining certain features of the process of alcoholic deterioration.

Conclusions.

The authors of both of the studies outlined above suggest that because of the presence of a constricted future time perspective among alcoholics in treatment, approaches that emphasize long-term changes and future negative eventualities are less likely to be viable with alcoholics than are treatment strategies that focus on immediate goals. Alcoholics Anonymous is one such treatment modality,with its emphasis on living one day at a time.

Although the time perspective results have been discussed as if they applied to all alcoholics, it would be of value to determine whether heterogeneity within this population has implications for treatment. In particular, it would be useful to know whether alcoholics with more extensive and coherent future perspectives stay longer in therapy, participate more in therapy activities, and carry a better long-term prognosis. In terms of augmenting treatment effectiveness, it would also be worth determining whether different treatment approaches are optimal for alcoholics with differing time perspectives. These questions do not appear to have been addressed in the literature.

Another question that does not seem to have been considered among alcoholics is whether time perspective alters over the course of therapy. It is unfortunate that the studies discussed above failed not only to consider this possibility but also omitted reference to when, during the treatment period, testing took place. It could be, as suggested by Landau (1976) from a study of prisoners and military conscripts, that institutionalization per se may in part account for decreased salience of future events. In his study, prisoners, as they approached the end of their sentence, showed an increase in future orientation relative to the past and present. To settle some of these questions, it would be useful to conduct a longitudinal study with alcoholics. In particular, it would allow some estimation of the extent to which a

constriction of future extension is a function of hospitalization as opposed to being a relatively stable trait. If patients were followed after release from therapy, it would also afford quantification of the extent to which changes during therapy (if they occur) relate to long-term outcome.

Religiosity.

Religious beliefs and practices have been little investigated in relation to therapy participation and outcome among alcoholics. This is somewhat surprising given the well-documented association between religious background and the incidence of alcohol problems. For example, Jews in many cultures are known to have very low alcoholism rates compared with Christian religions. This difference persists when occupational and educational variables are controlled. Additionally, in New Zealand, there are data suggesting that Roman Catholics are disproportionately represented in treatment settings. They make up approximately 15 percent of the country's adult population and constitute approximately 33 percent of alcoholism admissions (Gregson, 1978).

This neglect is also surprising given the widespread popularity of Alcoholics Anonymous as a treatment approach. Although it is frequently claimed by AA supporters that this movement is nonsectarian, and that the "Higher Power" to which members must acknowledge subservience is

not necessarily the God of the Judæo-Christian tradition, from the author's observations of AA education and meetings, the word God is used frequently and the Lord's Prayer is sometimes recited.[1] Other writers (e.g. Mowrer, 1965; Jones, 1970; Oden, 1972) have claimed that many of the AA doctrines have close parallels with Christian teachings. Whitley (1977), who is both a Theologian and a Sociologist, closely studied AA groups in the USA. In terms of the way in which these groups operate, he concluded "AA is a parareligious group, utilizing identifiable religious resources in the effort to achieve its goals." He further commented, "The paradigm for the group life and subculture of AA was, I found, remarkably similar to that of a Methodist class meeting...".

Some studies investigating treatment participation and outcome have included variables indexing religiosity In the main, the measures used have been coarse and the results have failed to implicate religious factors in either of these areas. For example, Kissin et al (1970) studied predictors of both premature therapy termination

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1. One ardent AA supporter informed the author that the "Higher Power" could even be a light bulb! This may be so. However, when asked if he had ever known anybody who construed it this way, he said that he had not. A few patients going through treatment programmes with which the author has been involved, have claimed to view the "Higher Power" as a symbol of the AA group, the AA movement as a whole, or as mankind. However, for the majority, the "Higher Power" does appear to be seen as God and their AA experience is frequently self described as "finding God" or as a "return to God".

and therapy outcome one year after discharge in four different types of treatment programme. Religion, defined by a dichotomous Protestant - NonProtestant categorization, failed to predict significant differences on any of the dependent variables. A more satisfactory range of religiosity measures was included in a treatment outcome study by Bateman and Peterson (1971). They used religious denomination, church membership, and self ratings of frequency of church attendance during the year prior to admission. None of these variables was found to discriminate significantly between abstinent and relapsed expatients six months after discharge from therapy. The relationship between these measures and premature therapy termination was not investigated.

Research on therapy participation and outcome appears to have been based on the assumption that both therapy and patient groups are homogeneous entities. More recently however, it has been shown that differences between therapy settings are important in determining dropout rates. For example, Smart and Gray (1978) found that programmes that included a variety of medical as opposed to nonmedical interventions tended to retain more patients for the full treatment period.

In describing predictors of relapse and premature termination earlier in this thesis, it was evident that the literature discussed did imply that failures and dropouts tended to be a single type of individual.

There is some support for this view. That is to say, some patient characteristics predispose individuals to a poor outcome in a wide range of therapy programmes - of the range currently available. In addition, some characteristics of treatment programmes appear to have a similar effect on a wide variety of alcoholics. However, there is some evidence that certain predictors are only significant in some types of programme. This was a major finding of Bateman and Petersons' (1971) study discussed above.

Research by Fiester and Rudestam (1975) with general psychiatric outpatients also supports the view that different factors contribute to dropout in different settings. Full understanding of this phenomenon requires recognition of both patient and treatment heterogeneity and a detailed investigation of the interaction between the two that occurs during the therapy process. Moos (Note 7) claims that such treatment-patient interactions and process variables more generally, have been largely ignored in the alcoholism literature.

Returning to Fiester and Rudestams' study for a moment. These authors concluded that because there were no differences between patients in the two outpatient settings that they evaluated (e.g. in terms of age, sex, and social class), the differences in the dropout patterns appeared to be primarily due to a divergence in therapist characteristics and therapy approaches.

From the above considerations, it seems that three groups of factors need to be considered when we attempt to predict therapy participation and outcome. First, there are those subject characteristics that affect outcome in a wide range of treatment settings (possibly certain demographic variables, sociopathy and perhaps depression, and some of the cognitive 'process' variables as defined in this thesis). Second, there are treatment variables that have similar effects on a wide range of patients (possibly the inclusion of medical as opposed to entirely nonmedical interventions and therapist qualities such as warmth, empathy and genuineness as defined by Rogers and others). Finally, there are other measures (it could be that some of the indices described in this thesis as cognitive 'content' variables, e.g. time perspective and religious ideology, are particularly relevant here) that interact with certain treatment variables (e.g. the ideological orientation of the treatment programme and the theoretical model held by therapists).

The above model allows us to make the prediction that if a wider variety of treatment approaches become available, then we could expect to find a relative increase in the number of measures that fall into the third category. This assumes that patients are randomly assigned, or randomly assign themselves, to treatment programmes. If, on the other hand, patients were matched to treatments tailored to their therapy needs, then within-programme variance, and subsequently the predicitive power of variables

in groups one and three, would decrease. This is expected to be a desirable outcome because it is assumed that it would accompany a higher retention rate and superior treatment record.

This brief digression from the specific topic of religion sets the scene for the discussion of a study that found religiosity to be a strong predictor of post-treatment abstinence.

Gregson and Taylor (1977), in a study described in some detail in the previous chapter, investigated the relationship of a wide range of independent variables to treatment outcome. A number of these variables were incorporated into two stepwise linear regression analyses that used time to relapse within the first six months after leaving hospital as the dependent variable. Of the three significant predictors in the first regression analysis, a measure of cognitive dysfunction (PCIT) was the strongest predictor of relapse (20 percent of the total variance). This was followed by self reported frequency of religious attendances during the year prior to therapy (15 percent) and social status (8 percent). The second analysis differed from the first only by the inclusion of one additional variable, AA membership and attendance throughout the followup period (40 percent of the total sample) versus nonmembership during this period.

These analyses implicated both religious observations

and AA participation as highly significant and strong predictors of outcome in this particular treatment programme. The decrease in the percentage of overall variance accounted for by religious activities in the second analysis relative to the first, occurred because of the inclusion of AA membership, a measure with which it correlated highly. The authors interpreted this finding as an indication that an active religious background predisposes individuals to accept membership to AA. This is not unexpected, considering the description of AA given earlier in this section.

It appears likely that the importance of religion as a predictor of outcome in this study relative to its apparent nonimportance in other studies, may have arisen because of the emphasis placed upon AA participation. Gregson and Taylors' description of the programme included the following observations:

> All patients were given extensive reading material focussing on the AA philosophy and programmes, and were encouraged to accept total abstinence as the only realistic goal. Discussion of the case histories reported in AA literature was a core topic in all therapy. The patients often learned to describe and evaluate themselves in terms of the AA Twelve Step programme. (p. 1751)

Elsewhere, they note that Priests and Lay Therapists (themselves AA members) were prominent among the primary therapists and that the programme presented AA as the best possible way of helping released patients to control their drinking problem.

This interpretation of the role of religion in the mediation of treatment outcome receives some corroboration from Bateman and Petersons' (1971) study referenced above. This study used a similar group of alcoholic patients. It also employed the same measure of religious attendance, the same followup interval, and a dichotomous abstinence/relapse outcome measure. The only major differences between the two studies appear to be the latter's much reduced encouragement of AA and its failure to demonstrate that religious attendance played a significant role in predicting outcome.

Like other studies, including those with a religious emphasis (e.g. Katz, 1966), Gregson and Taylor found that religious denomination was not a predictor of outcome. From this, they concluded that religious ideology was not as important in influencing outcome as was the social support component of active church attendance and AA membership.

The interpretation given in the last paragraph does not preclude the possibility that certain aspects of religious ideology influence alcoholics' predispositions to accept religiously oriented therapy or AA membership. Indeed, Gregson (1978) has subsequently suggested that the closer a patient's overlearned philosophical orientation is to that of a given treatment programme, the more likely it is that the patient will accept the treatment ideology and participate fully in therapy.

He refers to this hypothesis as 'ideological exchangeability' and argues that this is a necessary but not sufficient condition for treatment success.

In conclusion, it appears that religion can be an important factor determining treatment participation (although to date this has not been directly assessed) and in influencing treatment outcome. It may be however, that individual differences along religiosity dimensions are only important when the treatment setting has a religious orientation.

It is apparent from Gregson and Taylors' study, that religiosity can be added to the list of factors which have been shown to predict successful affiliation with AA. The others that have been most consistently corroborated include: a high need for affiliation, high authoritarianism, proneness to guilt, and a middle class background (see Trice & Raven,1970, for a review). Because many alcoholics do not fit this description, it could be predicted that programmes that emphasize AA will inhibit recovery in some patients. Gregson (1978) points out that the category of patients that is ideologically mismatched with AA forms an increasing proportion of alcoholics in treatment. Many alcoholics in this group, particularly among the younger acting-out members,do not meet some of the other predictive criteria as well.

Investigation of the questions raised in this area

should prove fruitful, both in advancing our understanding of the therapy process and in enhancing its effectiveness.

Conclusions.

This chapter has identified a number of cognitive variables which have been studied to varying degrees in relation to alcoholism. They share the distinction of having been ignored in major reviews of both the personality and the cognitive alcoholism literature. Along with measures of cognitive dysfunction, they hold the potential of accounting for a segment of the dependent variable variance that has to date been unexplained in attempts to predict treatment involvement and outcome.

Because these constructs are linked more to the content of cognition than they are to style or efficiency of information processing, they could be expected to interact more with the content and orientation of particular therapies. Consequently, they may only modify response to therapy in particular settings. For this reason, it will probably be more valuable to consider their operation within a person x treatment interactionist framework rather than concentrating on main effects. From this angle, a failure to predict outcome with one or more of these person variables does not warrant just a missing asterisk in a reviewer's tally table and the conclusion that this variable has not been consistently confirmed as a significant predictor. To the contrary, when treatment type is also brought into the picture, the

missing asterisk can be a very important piece of information.

At this point, no effort is made to draw the areas discussed together or to link them systematically with the literature pertaining to brain damage, cognitive dysfunction, or personality. Nevertheless, the author is struck by certain parallels between the time perspective impairments apparently found in many alcoholics and the neuropsychological sequelae of frontal lobe damage. Although tempting to pursue this and other leads, such an excursion into grand theory building would be largely based on inference and speculation. Given the shortcomings of the current literature and, to be honest, the state of our 'art' more generally, the exercise would not only be premature, it would be presumptuous.

CHAPTER FIVE

AIMS OF THIS STUDY AND RESEARCH DESIGN

General Aims.

Although a wide range of socio-demographic and psychological measures have been investigated with a view to building predictive models of therapy participation and outcome, cognitive factors of both the 'process' and 'content' type have been little considered in this respect.

Research in this area has often been haphazardly conceptualized. Measures are frequently included without a strong theoretical rationale and typically, efforts are not made to relate them conceptually to process variables pertaining to the treatment setting or the post-treatment environment. Investigations of psychological measures have often been confined to the consideration of just one construct or a closely related group of constructs.

Exercises of the type described in the preceding paragraph are of limited value in a complex multivariate situation. Omission of variables would not in itself be so important if we could assume that the omitted variables were relatively small contributors to the outcome under study and that, more significantly, they were independent of the dependent and independent variables in question. However, from the research reviewed up to this point, it has been suggested that some of these neglected variables,

for example cognitive dysfunction, have strong effects on outcome measures. For a start, this means that their omission will reduce the predicitive power of any framework that we build. This will in turn limit our understanding of treatment and relapse phenomena. Further, the literature brought together in the earlier chapters suggests that a number of the independent variables that have been related to outcome are interrelated in multiple and complex ways. Consequently, we cannot simply disregard variables in this category and construe them as background noise that can safely be ignored or left as part of the 'random variation' in an error term.

To some extent, we are never likely to be able to resolve the problems raised above. We will be unlikely ever to know all of the factors operating in a given situation let alone the details of interrelationships between these factors. This is a fact of life of applied psychology and we would be deceiving ourselves to think otherwise. However, we can obtain a more adequate grasp of the complicated dynamics of the treatment world if we at least include those measures that research and informed 'common sense' suggest are important. We can get even closer still if we include these measures in a multivariate design so that their interrelationships can be explicated.

The major aim of this thesis is to investigate the role of cognitive dysfunction in relation to indices of treatment participation and treatment outcome. In large

part, this concern grows out of the finding by Gregson and Taylor (1977) that this neglected area of psychological functioning may account for a large section of the unexplained dependent variable variance apparent in research in this field. It also arises from the conclusions reached earlier regarding the extent and type of cognitive impairment found among alcoholics, and its probable implications for the adjustments required in both the treatment and post-treatment environment.

The second major aim, that of evaluating the role of cognitive 'content' variables in relation to treatment, also in part arises from Gregson and Taylors' findings. In particular, there is a concern to investigate further the possible relationship of religiosity to treatment acceptance, involvement, and outcome within an AA-oriented therapy programme. Other 'content' variables, particularly locus of control and time perspective, are also studied in relation to the same dependent variables. Their inclusion is based on literature suggesting their relevance to treatment processes and post-treatment demands. Their inclusion also stems from the desire to identify other outcome-relevant dimensions.

In part then, one aim of the current study is an attempt to replicate Gregson and Taylors' major findings in another treatment population. This is a necessary undertaking for two reasons. First, their results suggest the operation of two groups of measures that other studies

have either ignored or have failed to implicate as strong predictors of treatment outcome. If Gregson and Taylors' results could be corroborated, then we will have advanced our knowledge of individual differences that influence response to therapy. Second, although related to the first consideration, a major form of analysis employed in their study was step-wise regression. This technique capitalizes upon chance variations in data. Consequently, it has been cautioned by Cohen and Cohen (1975) that if results "are to be substantively interpreted, a cross-validation of the step-wise analysis in a new sample needs to be undertaken, and only those conclusions which hold for both samples should be drawn" (p. 104).

The present study goes beyond Gregson and Taylors' work by incorporating additional cognitive and background measures, by considering change over time in therapy on some of these dimensions, by expanding the range of dependent variables to include indices of treatment participation and progress, and by extending the follow-up period from six to 12 months.

A further concern of the present research is to subdivide the 'relapsed' patients into those who have a reduced, controlled intake, as opposed to those who have a heavy 'alcoholic' intake. This categorization is made because of the pressing need to find characteristics that predict successful maintenance of a controlled drinking

outcome distinct from those that predict abstinence or relapse (see Smart, 1978, for a discussion of this issue).

Work in the field of alcohol studies generally has ignored gender differences. Carver (1977), among others, has argued that this neglect is a form of gender discrimination and that it is partly responsible for the failure to identify and take account of the particular treatment needs of women alcoholics. Certainly, with regard to the majority of psychological constructs discussed in the earlier chapters, little is known of gender differences, let alone how gender might interact with these factors in the mediation of treatment outcome. Although not a major focus of the present study, some attention is given to this issue.

More generally, by incorporating a wide range of measures within appropriate multivariate analyses, an attempt is made in the research conducted here to assess their importance, relative to oneanother, in relation to treatment participation and outcome. This is viewed as an important part of the research. It is not enough just to know that one category of patients is significantly different from another category on a given measure. It is also necessary to have some idea of the strength of the relationship - both with the outcome measure and relative to other independent variables. When clinical significance is seen as an important consideration, as opposed to getting a paper published, these questions

figure prominantly. Because the analyses that allow us to address this type of question include a description of the way multiple independent variables interrelate, they facilitate extension of the 'nomological network' (Cronbach & Meehl, 1973) of information pertaining to treatment and its relation to psychological processes. Alternatively, this can be conceptualized as an extension of the construct validity of the tests incorporated within the multivariate data structure. For this reason, the Booklet Rod-and-Frame Test (BRF; see Appendix 1 and 2), a test still in its development stage, was included in this study.

A final aim is to increase our knowledge of the drinking-related variables that are antecedent correlates of cognitive dysfunction found among alcoholics during treatment.

A Systems Model of Cognitive Functioning, Treatment Involvement, and Treatment Outcome.

There are a large number of questions addressed in this thesis, questions which involve the consideration of many variables sampled at different points in time. To provide an overall structure within which these questions can be meaningfully discussed, a systems model of therapy participation and outcome is outlined, making explicit a number of the author's major assumptions regarding these processes and the direction in which

causal influences are expected to run. By systematically decomposing this model, each of the major areas of concern can be identified without losing sight of their position within the overall system.

In describing the systems model, the major findings of the literature reviewed earlier are placed into a broader perspective. From this description, the major hypotheses of the present study emerge, along with an account of the independent and dependent variables that are relevant to these hypotheses. Consequently, this model provides a structure for the subsequent sections of this thesis: the hypptheses, research design, results, and discussion.

The model is outlined in Figure 1. As already stated, the framework summarizes at a general level, the major causal interrrelationships between sets of variables related to cognitive state, treatment participation, and treatment outcome. The boxes are processes in time or points within the nomological network[1] that have observable indicators. In other

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1. Meehl (1978) uses the term 'nomological network' to designate "the system of lawlike relationships conjectured to hold between theoretical entitites (states, structures, events, dispositions) and between theoretical entities and their observable indicators. The 'network' metaphor is chosen to emphasize the structure of such systems, in which the nodes of the network, representing the postulated theoretical entities, are connected by the strands of the network, representing the lawful relationships hypothesized to hold between

words, they are regions within the theoretical system where we can make measurements with some degree of accuracy. There are other areas where such observations cannot be made. These areas derive their meaning from their relationship to the overall conceptual structure, and only connect indirectly with an observational data base. The direction of the causal links (our theoretical postulates) are indicated by arrows and are labelled in accordance with standard path analysis conventions.

Although portrayed as a closed system, it is recognized that this is an oversimplification. A number of potentially significant variables are not included in the model as it stands. Indeed, each of the boxes could be visualized as having additional arrows entering them from outside of the system, representing causal factors not explicitly covered in the present model. Some of these extrinsic factors could be expected to interact with the variables included in the model. Consequently, it is necessary to consider some of the more likely of these 'nuisance variables' and include them in some of the analyses. Examples of this type of variable include age and gender although, because both of these characteristics carry social meaning and expectations, they could in part be represented in box 4 of the system model.

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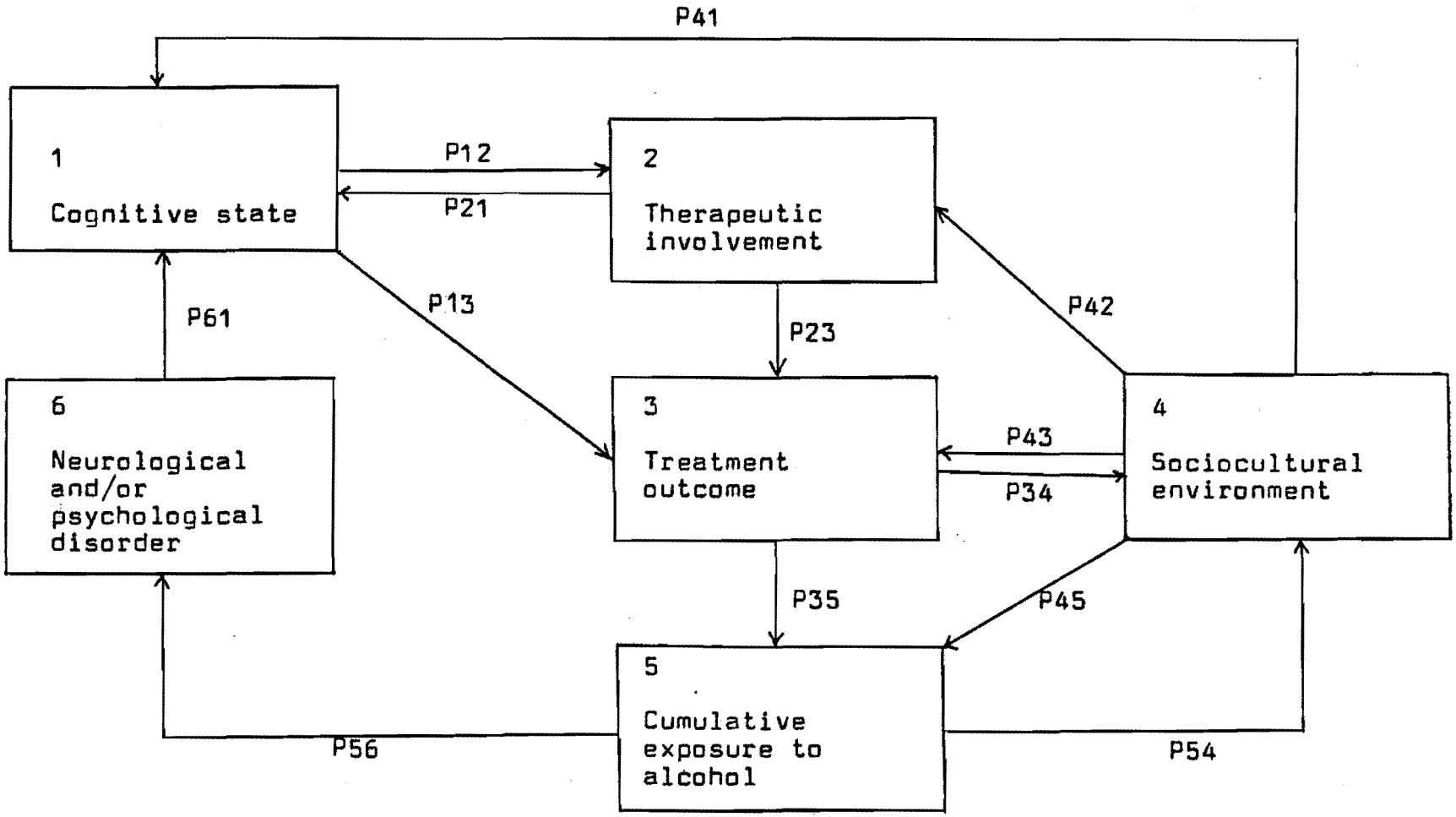
1 ctd. the entities." The set of theoretical statements can be regarded as a system rather than a collection of unrelated assertions because of the input-output relations that link the nodes.

Figure 1: A system model of cognitive functioning, therapy involvement, and treatment outcome.



Box 1 of the systems model portrayed in Figure 1 includes cognitive measures of both the 'process' and 'content' type as described in chapters 2, 3, and 4, and as assessed during hospitalization. Paths 61, 41, and 21 impinge upon this category of measures.

Path 61 acknowledges the research reviewed earlier that suggests prolonged exposure to excessive alcohol intake produces deficits in cognitive efficiency, for example, as indexed by measures of fluid intelligence and neuropsychological tests. It has yet to be established whether or not alcohol consumption or drinking-related experiences lead to changes in the content area although it would seem probable that external scores on the Drinking-related Locus of Control Scale and constricted future extension are a consequence of such events. External scores on measures of generalized locus of control may also, at least in part, arise in this way.

Path 41 recognizes that sociocultural factors also have some effect on cognitive performance. Such effects related to gender, age, and socioeconomic status are of major concern in neuropsychology where attempts are made to infer the organic condition of the brain from test performance. Religious ideology and practice is expected to be very strongly influenced by processes represented in Box 4 and it is likely that performance on other 'content' measures are also partly influenced from this source.

Path 21 is a reflection of the demonstration that changes in cognitive state can occur during the post-detoxification period and that some of these changes (e.g. decrease in RFT field dependence) are probably influenced by the treatment environment per se.

There is some suggestion from a variety of empirical studies that factors not included in the model may also be determinants of cognitive state. Both the work on childhood minimal brain dysfunction and some studies related to RFT performance suggest that some cognitive dysfunction antedates the development of alcohol problems in a subgroup of alcoholics. However, irrespective of the origin of cognitive impairment recorded during hospitalization, there is a little empirical evidence, and strong theoretical reasons, for believing that its presence has important implications for processes occurring in Box 2.

Box 2 refers to patient involvement in therapy activities. This can be assessed in a variety of ways including: whether or not patients stay in treatment as opposed to leaving against advice to the contrary, the degree to which they engage in therapy activities, ratings of improvement by either staff or patients themselves, or changes on any of a variety of psychological measures.

Path 12 formalizes the hypothesized relationships between measures of cognitive state and indices of therapy involvement. It is predicted that a variety of measures

of cognitive state will compromise improvement in this area. Some of this effect may be mediated via learned avoidance responses linked to the emotional sequelae of decreased cognitive efficiency. The strongest predictors are expected to be degree of cognitive integrity and degree of patient-therapy ideology match - in the case of the programme under study, religious ideology.

Path 42 reflects the view that factors from the sociocultural environment will also modify response to therapy. An example here is whether or not a patient has an intact social network, including a stable marital relationship. This could have a motivating effect during therapy. Socioeconomic level could also be important for a variety of reasons, one being the probability that higher socioeconomic level is linked with higher verbal facility and increased commonalities generally with the treatment programme and its staff.

It is predicted that therapeutic involvement and change (e.g. increased insight) bears some relationship to treatment outcome, although it is also expected that such effects will dissipate as a function of time as other life events intervene. This causal link is represented by p23. Although largely neglected in the literature, it is also expected that post-hospitalization experience within the ex-patient's socio-cultural environment will also have a strong influence on treatment outcome. For example, the patient who returns to an

intact marriage and a job that does not make excessive demands on his or her adaptive abilities is likely to carry a better prognosis. There is likely to be an interaction effect here however (p34), where initial outcome influences the feedback that the ex-patient subsequently recieves from the post-treatment environment. For example, whether or not the ex-patient is employed or joins AA could be expected to have a powerful effect upon the environmental experiences and reinforcement contingencies that impinge upon that individual.

Treatment outcome (which includes but does not consist only of whether or not the ex-patient relapses to a heavy alcohol intake, abstains, or stabilizes at a lower controlled level of alcohol consumption) is, of course, directly related to Box 5, cumulative alcohol intake. Continued high level alcohol consumption is expected to have disruptive effects on the immediate social environment of the ex-patient (p54). This could involve loss of job, rejection from AA members, and/or marital breakup, among other possibilities. This in turn could modify the individual's psychological state and social milieu in a way that could be expected to reinforce a pattern of excessive alcohol intake (p43 and p45). Continued alcohol abuse and associated experiences (e.g. dietary neglect, fights, accident-related head injuries) are likely to produce further neurological and psychological disorders (e.g. depression, impulsive antisocial behaviours) (p56) that are also partly responsible for further cognitive

impairment and attitude changes (e.g. feelings of loss of control over life events) (p61).

Path 13 is included because cognitive state assessed during hospitalization (particularly if tested near the end of the treatment period), is likely to persist into the post-hospitalization period and continue to exert an effect on attempts to cope with environmental demands. As mentioned earlier, this area has been little studied.

Apart from summarizing the hypothesized causal interrelationships between sets of variables related to treatment participation and outcome, this model also describes the reasoning behind the order in which certain variables are sequentially incorporated into later regression analyses. This sequential character of the operation of major sets of variables is important for inferring causal relationships related to treatment participation and outcome. The reason for this being that time-lagged correlations provide a stronger basis for causal inference than is the case with correlations where the measures are assessed cross-sectionally. Indeed, the latter are virtually useless in this respect.

The model also enables the derivation of both direct and indirect paths through which different measures can affect and be affected by other groups of variables. For example, cognitive state not only has a direct effect on treatment outcome (p13), it also has an indirect effect

via interactions with other variables, such as treatment involvement (p12p23). By considering both direct and indirect effects, a greater understanding of the phenomena in question is afforded the investigator.

The major hypotheses addressed in this thesis have been described in the above account of the systems model. They are stated in a more specific form at the end of this chapter, after the particular measures employed in the current study have been described.

The relationship of the present work to the previous Gregson and Taylor study is more clearly stated by a consideration of the systems model. These writers showed a relationship between PCIT (a measure of cognitive dysfunction) representing Box 1, and treatment outcome (indexed by time to relapse) (p13). They also strongly implicated post-treatment AA membership (one of many possible measures from Box 4) in treatment outcome (p43). The present study differs by sampling measures from a wider variety of points in the system and by testing a number of additional relationships, represented by the causal paths specified within the model.

Method

Subjects

One hundred and six chronic alcoholics, comprising 74 men (mean age 42.0, SD = 13.7) and 32 women (mean age

46.7, SD = 12.4), were interviewed and assessed during an 8 - 10 week in-patient treatment programme for alcoholism. All had a primary diagnosis of Alcoholism Addiction. Although some were given a secondary diagnosis of neurosis or personality disorder by admission staff, none carried a psychotic diagnosis. Although neurological examinations were not conducted, none of the patients were considered by Medical staff to evidence sufficient clinical signs to warrent one of the chronic alcoholic brain syndrome diagnoses.

Patients were referred by a variety of Health Professionals from many parts of New Zealand. Some patients had previously had a period of detoxification (generally between 1 to 2 weeks although in a few instances longer) prior to admission. Others had been drinking up until or near to the date of admission.

Eleven percent were from professional and administrative occupations, 30 percent from clerical and skilled work, and 37 percent from semi-skilled and unskilled work. Fourteen percent gave housewife as their occupation (48 percent of the women). The remaining women are included in the occupational percentages. Six percent were unemployed and eight percent either retired or disabled.

The following percentages were married (50 percent), separated or divorced (20 percent), widowed (6 percent), and never married (24 percent). Five were Maori, the remaining 101 of European ancestry.

From patient self-reports, the average daily intake of alcohol was an equivalent of 218 (SD 115) grams of ethanol. The mean number of years prior to admission during which patients considered alcohol to be a problem in their lives (of 'alcoholic proportion') was 10.4 (SD 8.8). The mean number of previous admissions to programmes for alcoholism treatment was 0.6 (SD 1.0).

The subjects comprised successive admissions to Queen Mary Hospital, Hanmer Springs, New Zealand, from mid April to mid July 1977, and from mid February to the end of March, 1978. The 106 included in the main part of the present study were those who completed the programme. During the testing periods, 28 further patients left against medical advice and prior to completing all of the psychological tests. A further six patients completed the course during this time but were not included. One refused to participate. Another was mentally subnormal, illiterate, and unable to comply with the test instructions. The remainder were either on leave or too ill physically at the time all or part of their testing was planned to take place.

Treatment Programme.

The structure and content of the hospital's treatment programme has been described elsewhere (Gregson & Taylor, 1977). Some mention of it is also made in Chapter 4. Since the Gregson and Taylor study however, some additional

components have been incorporated into the programme. These additions included transactional analysis, psychodrama, and grief groups. Nevertheless, the overall structure of the programme, resting strongly on a group therapy and lecture format emphasizing AA philosophy and abstinence as the only viable treatment goal, remained. Another change in the programme was a reduction from a fixed 12 weeks of inpatient treatment to an eight to 10 week stay. Additional flexibility was added by the inclusion of a separate two-week programme for multiple recidivists. Patients in this group were not included in the present study.

Data: Measures and Collection Procedure.

Data consisted of (a) a background information form administered individually during an initial interview shortly after admission to the programme; (b) information on longer-term drinking history elicited during the initial interview; (c) a booklet version of the Rod-and-Frame Test (BRFT) individually administered during the initial interview and repeated five to six weeks later; (d) the Patterned Cognitive Impairment Test (PCIT), individually administered at the time of the second BRFT assessment; (e) standard psychological questionnaires, group administered in a randomized order within a day or two of both BRFT administrations; (f) Insight ratings completed by primary therapists at the end of the first two weeks of treatment and again at the end of the programme; (g)

a Treatment Experiences Form completed by staff at the end of the programme; (h) Follow-up Information Questionnaires mailed to ex-patients three and 12 months after they left the programme; (i) Brief Followup Questionnaires mailed to referees and Health Professionals at the same time questionnaires were sent to ex-patients.

Initial Interview. The author spent approximately 90 minutes with each patient some time during their first 18 days of hospitalization. At this time, a rationale for the research was given (i.e. to increase our understanding of how treatment works and how we might, in the future, be able to increase treatment effectiveness by tailoring it to the needs of specific patient groups) and the patient's participation sought. At this time, the patient was asked for the name of two referees who could give an impartial account of their subsequent post-hospitalization drinking behaviour and general well-being. In some cases, one of the referees nominated was a family member. The patient's permission was also sought to consult their General Practioner and/or the referring Health Professional. Patients were also questioned about their drinking history to elicit information concerning how long they considered drinking to be a problem and to obtain a rough index of their total consumption over the years. It was explained to each patient that any information given and all test results were strictly confidential, that nothing would be passed on to hospital staff, and that the author was attached to a University, not to the hospital.

During the initial interview, each patient was administered the BRFT (see Appendices 1 and 2). Following this, they were given the Background Information Form to complete. This form (see Appendix 3), elicited biographical details, social data, information on drinking behaviour, a self-rating of the severity of drinking problems, and attitudes towards Alcoholics Anonymous. It also included scales measuring alcohol-related physical and psychological symptoms (e.g. vomiting, blackouts), psychological functioning (e.g. anxiety, depression symptoms), and social functioning (e.g. attendance at social functions, social interests) for the month prior to admission.

Patterned Cognitive Impairment Test (PCIT). This test battery was briefly described earlier (p. 157). Fuller accounts are found in Gregson and Taylor (1977) and in the test manual (Gregson & Abbott, 1979). PCIT was administered individually to patients by the author during their seventh or eighth week of treatment. BRFT was also readministered at this time. The order in which the two tests were administered was randomized with a fifteen minute gap between the two administrations.

Psychological Questionnaires. These were administered to groups of six to 12 patients. On both test occasions, individuals comprising the groups had, within the preceding two days, been tested individually with BRFT or BRFT and PCIT. At the individual assessments, a careful note was made of whether or not patients were likely to require

assistance in reading the questionnaire instructions and items. Any apparent anxiety was discussed and ways of assisting considered. In most instances, patients in this category said that they would not mind asking for assistance if the need arose during the group testing. Three patients claimed that they would be embarrassed if this procedure was followed. In these instances, the author saw the respondents individually and read the items aloud. Patients indicated their responses on another copy of the questionnaire being presented.

The following questionnaires were administered:

1. Locus of Control (Rotter, 1966). This questionnaire was discussed in some detail above (pp. 172 - 174). It is included in Appendix 4.
2. Alcohol-Related Locus of Control (Note 3). This questionnaire and related research was discussed in Chapter 4, pages 188 - 191. Only the items loading on the three main factors were included (see Appendix 5).
3. Time Reference Inventory (TRI) (Roos, 1964). A short form of this test was used (see Appendix 6). Roos and Albers (1965) consider that this measure yields equivalent results to their longer original. The present form was modified slightly by Black (1969) to make it conform with New Zealand idiom. The test was included partly because the TRI had previously been used with alcoholics (see Chapter 4) and because it gives both a standardized measure of past extension and a temporal orientation score. The last of these two measures is unique to this test. It also yields a future extension score (the mean of the

difference between the chronological age of the subject at his or her last birthday and age responses given to 'future' items). The past extension score is derived in an analogous manner. The temporal orientation score is obtained by summing the age differences over all items and divided by 12, the number of questionnaire items.

4. Future Events Test (FET) (Stein & Craik, 1965). Twenty-five events are listed (see Appendix 7). The subject is required to select any 10 and state how old he or she expects to be when each occurs. The future extension score is the median of the 10 lengths of time (age given for each event minus present chronological age). This scoring method was suggested by Siegman (1961) and has an advantage over alternative methods (e.g. Wallace, 1956) in that it reduces the likelihood of chance differences arising between subjects.

5. Religiosity Questionnaire. This questionnaire was developed by Rawlings (1971) to measure religious beliefs and the degree to which values are shaped by Christian or non-Christian beliefs. The items included in the questionnaire used in this study were those loading on the two main factors identified by Rawlings. The first factor ('G1') is defined as strength of committment to Christian dogma and is derived by adding scores on items 1, 2, 3, 6, 8, and 15 of the questionnaire included in Appendix 9. The second factor ('G2') score is obtained by adding items 4, 10, 11, and 14, and by subtracting 5, 7, 9, 12, and 13. A high score on this factor reflects an attitude towards behaviour that is "based on rational

explaining which patient behaviours led them to give their particular rating. Large disagreements were evident in only one of four interviews. It was subsequently found that this occurred because some staff were taking into account information that they had on the particular patient from outside of both the video and hospital setting. In the final interview, 11 of the 14 staff were in agreement. The other three were one point to either side of the modal score.

Insight ratings for each patient in the study were made by primary group therapists at the end of the first two weeks of hospitalization and again for the final two weeks of each patient's stay. In some instances, independent ratings were collected from co-therapists as a further reliability check.

Treatment Participation. At the end of each patient's hospital stay, treatment participation forms were completed by the primary therapist and nursing staff who were most familiar with the patient in question. A copy of this form is given in Appendix 10.

Followup Questionnaires. Three months after each patient left treatment, the author posted a questionnaire to that patient. The questionnaire and covering letter is included in Appendix 11. The questionnaire content is similar to the Background Information Form described above. If no reply was received by three weeks, a second copy of the questionnaire was sent. This procedure was repeated at

considerations uninfluenced by religious beliefs" and a low score reflects the "degree to which attitudes towards conduct are a reflection of Christian faith and belief" (Rawlings, 1971, p. 18). This questionnaire was included because a measure of committment to religious ideology was sought. Although adequate reliability data are not available for this questionnaire, this defect was offset by normative data from fundamentalist religious groups through to humanists and rationalists. Thus, suitable normative information from New Zealand samples was available for comparison with the present alcoholic population. This questionnaire is included in Appendix 8.

Insight Ratings. The insight rating form had been used previously by hospital staff. This measure was included because of this familiarity and because staff considered that the rating guide (see Appendix 9) reflected in a meaningful way a major aim of their insight-oriented treatment programme. To assess the degree of reliability pertaining to this measure when used by staff, all therapists who would be using the scale in the present study were included in video-assisted training sessions.

Videotaped interviews with a variety of patients were shown to the staff and their individual ratings were sought. Initial inter-participant agreement was moderately high with all staff members giving ratings within a range of three points on the five-point scale. After each video interview, individual ratings were collected and displayed to staff. The ratings were then discussed with each rater

12 months.

At three and 12 months, a followup questionnaire and covering letter (see Appendix 12) was also mailed to both nominated referees and health professionals with whom a patient was likely to have had contact. The information sought was kept to a minimum to increase the probability of a response. Staff at the hospital were also consulted as a further cross-check. They often received relevant information from an informal network of colleagues, ex-patients, and the AA 'grapevine'.

Treatment Outcome Measures. Outcome data was derived from the information provided by the followup procedures just described. Although impossible to obtain detailed information on post-hospitalization alcohol consumption for all patients, a rating on a scale of none/slight/heavy/incapacitating for work, provided an adequate categorization. Where there was a contradiction in follow-up accounts, further investigation followed. When doubt still remained, the worst outcome was taken as valid if the difference was between referees. When referees indicated a poorer outcome than the patient's own account, the evaluation of the former was preferred. When the situation was reversed, the patient's account was considered valid. Thus the results, if biased, are likely to be biased in the direction of an overcautious or conservative outcome assessment.

As suggested earlier, outcome measures selected for therapy evaluation should reflect both the aims of the

programme and the theoretical model held by the investigator. In the present instance, although the therapy programme taught total abstinence as the only viable outcome and strongly encouraged AA membership, there is now considerable doubt regarding the validity of the disease model upon which this approach is predicated (see Chapter One). For some so-called 'alcoholics', controlled or reduced intake appears to be a viable alternative (e.g. Pattison et al, 1977).

In the present study, a three-way categorization of drinking outcome was employed in a number of the analyses. Abstinence was defined as no alcohol intake during the three months prior to followup. Controlled drinking was defined as a slight intake with the additional observation by at least one referee that this was not disrupting social or work performance, or, one or two relapse episodes followed by at least four continuous weeks of abstinence immediately preceding the followup date. The remaining ex-patients were placed in the 'relapsed' category even though some of these people were drinking less and coping better generally than they were before hospitalization. In addition to this tripartite categorization of drinking outcome, the interval in weeks from the time the patient left hospital to the time of their first drink was also used in some analyses.

Another outcome measure assessed in the present study was whether or not ex-patients held paid employment at the time of followup. This and more detailed

outcome measures covering social and psychological functioning were provided by the followup questionnaire sent to ex-patients. Although 79 percent of the patients who completed the programme returned useable data three months after discharge, neither this nor the similarly detailed 12-month outcome data (from 72 percent of the ex-patients) are included in the present investigation. These measures will however be addressed in a subsequent report.

Data Analysis

A number of the major independent variables outlined in this chapter and discussed in the literature reviews will be addressed in relation to appropriate dependent variable measures (by *t*-tests and contingency table analyses). They will also be considered in relation to oneanother in multivariate analyses, analyses which are also conducted to address some of the major questions outlined in the systems model of treatment participation and outcome. The particular analyses employed and the rationale behind them will be briefly discussed in the results section.

Major Hypotheses

The following hypotheses are generated from the theory and research findings outlined and discussed in earlier sections of this thesis, and brought together in the systems model:

1. The alcoholic sample will be more impaired cognitively (as measured by PCIT and BRFT) than nonalcoholic groups;
2. Severity of chronic alcohol consumption patterns and increased age will correlate inversely with PCIT performance;
3. Cognitive dysfunction (PCIT and BRFT) will be a strong predictor of post-treatment drinking outcome.

With the partial exception of generalized locus of control, the 'content' cognitive indices that are included in the present study have been little investigated in relation to alcoholism. There is also difficulty when attempts are made to relate theory and research findings that have accumulated around these constructs in areas other than alcoholism to therapy participation and outcome. This is because in the case of alcoholism, little is known of these processes. Because of the paucity of prior knowledge, a portion of the analysis must be regarded as exploratory. Nevertheless, some hypotheses are stated apriori, namely:

4. Severity of chronic alcohol consumption patterns and increased age will show a positive correlation with externality (on both locus of control measures) and a past-oriented temporal orientation;
5. From the cognitive measures administered at the start of treatment, a linear composite will be derived which will bear a statistically significant

predictive relationship with a linear composite of treatment participation indices. Between these two sets of measures, dominant causal paths will link religiosity to voluntary attendance of both religious services and AA meetings. Another dominant causal path is predicted to run from degree of cognitive impairment to insight level at the end of therapy;

6. Over the course of therapy, the subjects will become more internal on both measures of locus of control, more future-oriented on measures of time perspective, and will show a greater acceptance of religious dogma;
7. Changes of the type listed under hypothesis 6 are expected to relate to extent of treatment participation and degree of insight at the end of treatment. At the most general level, it is predicted that a linear sum of change scores on the cognitive tests will be identified which will show a significant multivariate correlation with a linear sum of treatment participation indices;
8. Intensity of treatment participation and higher levels of insight at the end of treatment will be associated with a more successful outcome following discharge from the therapy programme.

With regard to the relationship between locus of control during hospitalization and subsequent treatment outcome, extant theory suggests two possibilities, both of which will be evaluated. The two possibilities are:

9. Internality (as indexed by both IE and DRLC) will be associated with a successful post-treatment outcome;
10. Both extreme internality and extreme externality will be associated with relapse following discharge.

The locus of control literature reviewed earlier also suggests that:

11. Drinking-related locus of control will be a more powerful predictor of post-treatment drinking outcome than generalized (IE) locus of control.

Other cognitive 'content' measures are also expected to bear a predictive relationship to post-treatment outcome. In particular:

12. High religiosity as measured by both frequency of self-reported religious observance prior to hospitalization and acceptance of religious dogma during hospitalization, will be associated with successful post-treatment drinking outcome.
13. Future-oriented time perspective during treatment will carry with it a successful post-hospitalization prognosis.